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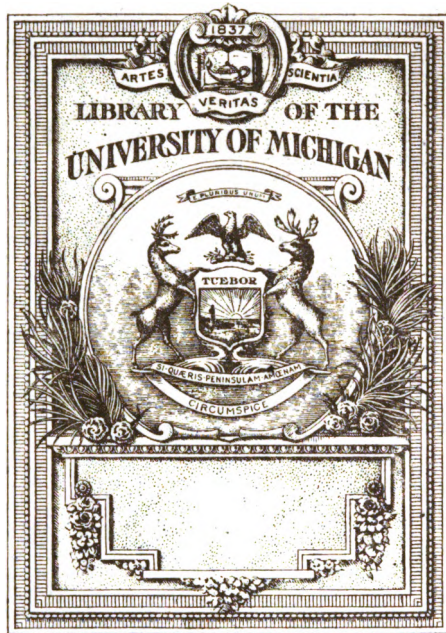
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WAR SURGERY
OF THE
NERVOUS SYSTEM



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1917

WAR SURGERY OF THE NERVOUS SYSTEM

A DIGEST OF THE IMPORTANT MEDICAL
JOURNALS AND BOOKS PUBLISHED DURING
THE EUROPEAN WAR

COMPILED BY THE DIVISION OF BRAIN SURGERY
SECTION OF SURGERY OF THE HEAD

U. S.
OFFICE OF THE SURGEON GENERAL'S OFFICE.
War Department : Washington, D. C., 1917



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PREFACE.

This manual represents an attempt to collect, digest, and arrange in orderly form, the literature of war surgery of the skull, brain, spine, spinal cord, meninges, and peripheral nerves, from August, 1914, to August, 1917. The sources of supply were the English, German, and French weekly, monthly, and quarterly medical journals, and those foreign treatises dealing with war surgery as practiced and observed during the present conflict.

The scheme adopted has been based largely on the plan of the collective abstract. It was thought wise to furnish fairly full abstracts, in order both to avoid unwarrantable dogmatism, and also in order to allow the reader free scope of personal interpretation. For this latter reason also, the editor has refrained both from extended critique and from attempting generalized conclusions, by way of summary.

No abstracts on the subject of Roentgenology have been furnished for the reason that special schools are equipping men for this work. The following are satisfactory references: A. G. Straw, *Arch. of Radiol. and Electrother.*, May, 1917, p. 393; W. Oram, *Arch. of Radiol. and Electrother.*, February, 1917, p. 277; H. E. Gamlem, *Arch. of Radiol. and Electrother.*, November, 1916, p. 175; Gage, *Arch. of Radiol. and Electrother.*, June, 1917, p. 1; E. Skinner, *Amer. Jour. Roent.*, June, 1917, p. 350; George H. Makins, *Brit. Jour. of Surg.*, June 16, 1917, p. 803.

Since the war hospitals may not be well supplied with books, we have introduced the abstracts with selections from standard textbooks, so that the reader of the manual might be always in close touch with fundamentals. For the brain, we have used the chapters from Keen's Surgery written by Dr. Harvey Cushing, and chapters from Dr. Isaac H. Jones's forthcoming book on Equilibrium and Vertigo. For the spine we have used selected chapters from Dr. C. A. Elsberg's book on Diseases of the Spinal Cord and its Membranes, and Dr. Charles H. Frazier's volume (in press) on Surgery of the Spine, and for peripheral nerves we have selected the chapter on peripheral nerves, written by Dr. Gordon M. Holmes for Osler's Modern Medicine.

The use of the phrase "war surgery" must be taken with a good deal of qualification, lest one fall into the error of thinking of this type of work as separate and distinct from the surgery of civil life.

As a matter of fact the surgical principles governing both are in large part exactly the same. The laws of ballistics, trench life, the terrain of the battle field, the problems of transport, and numerous other incidentals serve to modify established principles of surgery, but not more than that. Indeed nothing demonstrates more clearly the truth of this statement than the fact that war surgery makes such free use of those aids which, in civil surgery, are indispensable for both the laying down and following out of principles—bacteriology, serology, roentgenology, and the general routine of clinical microscopy.

Thanks are extended to the editor of Surgery Gynecology and Obstetrics for the permission to use abstracts from this journal. As a result of this much-appreciated courtesy we were able to complete an emergency task within a necessarily very short time limit. Thanks are also extended to J. B. Lippincott Co. for the privilege of quoting from Dr. Isaac H. Jones's forthcoming book, *Equilibrium and Vertigo*; to W. B. Saunders Co. for the privilege to use Dr. Harvey Cushing's contribution to *Surgery, Its Principles and Practice*, edited by William Williams Keen, M. D., and *Diseases of the Spinal Cord and Its Membranes* by Charles A. Elsberg, M. D.; to Lea & Febiger for the privilege to use Dr. Gordon Holmes's contribution to *Modern Medicine*, edited by Sir William Osler, M. D., and Thomas McCrae, M. D.; and finally, to D. Appleton & Co., for the privilege to use the chapter from Dr. Charles H. Frazier's forthcoming volume on *Diseases of the Spinal Cord*. We feel a deep sense of obligation to all these authors, from whom we have drawn so freely. It was unfortunately impossible to communicate directly with Dr. Cushing or with Dr. Holmes, in order to secure from them an expressed willingness, which we were sure they would accord us if time permitted.

CHAPTER I.

SURGERY OF THE HEAD.

(Parts 1-2 from Dr. HARVEY CUSHING's contribution to *Surgery, Its Principles and Practice*. Edited by W. W. Keen, M. D.; published by W. B. Saunders Company.)

PART 1.

FRACTURES OF THE SKULL.

Terminology.—The skull may be said to be fractured in distinction to its being wounded when, as the result of a blow, it becomes cracked or broken into more or less separate pieces. These injuries are classified in a variety of ways:

- (a) According to the mechanism of their production.
- (b) According to the presence or absence of a communicating wound.
- (c) According to the form assumed by the fragments.
- (d) According to their situation.

(a) Depending on the mechanical factors at work in their production, they are distinguished as (1) *bursting fractures* and (2) fractures due to local depression or indentation—so-called *bending fractures*. In the case of fractures the result of penetration by modern high-velocity projectiles a further mechanical element comes into play, producing *expansion fractures* through the explosive force of hydrodynamic action.

(b) Fractures are *open* or *compound* when they are exposed by a wound of the overlying soft parts; they are *simple* or *closed* when the soft tissues covering them remain intact.

(c) According to their form they are distinguished: As *linear* or *fissured* fractures when the bone is merely cracked without displacement; as a fracture by *diastasis* when there is a simple separation of the sutures; as *comminuted* or *fragmented* fractures when the lines of fracture intersect, so as to isolate separate particles of bone; as *depressed* fractures when fragments of bone, whether of the entire cranial thickness or of the inner table alone, are driven below their spherical level; as *perforating fractures* or *fractures with loss of sub-*

stance when they are the result of punctured wounds or when the fragments at the seat of the penetration have been carried away, leaving a defect, as is the case in most penetrating bullet wounds. And of these chief varieties many subdivisions may be made.

(d) Lastly, depending roughly on their anatomical situation, they are distinguished as *fractures of the base* and *fractures of the vault*, and although the two are often combined they may exist separately and have different characters. Thus, fractures of the base are usually linear and their fragments, if comminuted, are rarely displaced, for the base is much less accessible to direct injury; hence, fractures there are usually the indistinct result of violence applied elsewhere. On the other hand, the vault is directly exposed to injury and local comminution with dislocation of fragments is common, and as the bone is thick and has two determinable layers there are special influences which modify the character of the fragmentation.

(a) **The mechanism of fractures—Bending, bursting, and expansile fractures.**—Regarded as a hollow shell of bone which possesses elasticity sufficient to rebound when dropped, the cranium must needs differ from all other bones of the skeleton in the mechanism of its injuries. Certain of the physical laws which explain the peculiar form assumed by these injuries are known to us; others are still in dispute, and though, from a strictly clinical point of view, of chief importance is the knowledge that under certain conditions breaks occur in a certain manner and lead to certain complications, we naturally search for an explanation of the reason why they so occur, even though this information may in no wise affect our diagnosis, prognosis, or treatment.

Teevan, Wahl, Rauber, Félizet, Aran, Bohl, Bruns, Bergmann, Kocher, and a host of others have undertaken clinical and experimental investigations directed toward the elucidation of the underlying principles governing cranial fractures.

We must take into consideration the double effect of an impact, for the blow may produce (1) disturbances which are direct and chiefly of local consequence, and (2) those which are indirect and lead to solutions of continuity at a distance. Setting aside for the moment its irregularities and considering the skull to be an elastic globe, an impact will momentarily lessen its diameter in line of the blow, and force nearer together the point or pole of impact and the point on the sphere diametrically opposite. As the impact forces the poles together it will at the same time bulge out the sides of the sphere and thus increase the equatorial circumference and, in a lesser degree, the circumference of all the other circular planes which lie perpendicular to the polar diameter. If the distortion following the impact is inconsiderable the skull, owing to the elastic rebound, will resume its former shape unimpaired. If the distortion, on the other hand, is

so great as to overcome the molecular cohesion of the bony particles, they will be disrupted. This may take place (1) as a rupture or bursting of the bone in parts remote from the poles of impact where cranial dimensions have been increased to the point of overcoming tensile strength of the particles, and (2) as a local indentation at the pole of impact where cranial dimensions have been diminished to the point of overcoming the local resistance of the particles to pressure. These two qualities of elasticity—tensile strength and resistance to pressure—have been the objects of special study by Rauber, who has shown that resistance to pressure is a third greater than tensile strength. This, however, does not mean that fractures are less likely to occur at the pole of impact than at a distance, for other factors come into play.

Local character of injuries through bending.—These fractures usually result from the sharp impact of a body with a comparatively small surface. Such a blow expends its force quickly and a rebound occurs before the form of the skull, as a whole, has been sufficiently altered to produce lesions at a distance. At the pole of impact the bone is broken and the displaced fragments do not resume their former position.

In spite of its greater thickness and vaulted construction fractures of this sort are more common on the exposed calvarium than at the inaccessible base. The character of the lesion, furthermore, is influenced by the structural peculiarity of the bone; namely, its two dense tables separated by a spongy diploë. Owing to this, an indentation which leads to a bending fracture will cause the inner table to splinter and give way before the outer. In consequence we not infrequently find fractures limited to the inner table—a circumstance known even to the earliest writers in medicine, who explained the phenomenon on the supposition that the inner table was more fragile or brittle than the outer one, hence the “vitreous” surface. Not until Teevan’s studies was the process satisfactorily explained on the ground of tensile strength or cohesion of particles on the one hand and of resistance to pressure on the other. There is no simpler illustration than the oft-used one of a green stick broken across the knee. The cranial impact leads to a local indentation, which tends to pull apart the particles comprising the inner table and to drive together those of the outer. In certain rare cases the process may be reversed and the outer table alone suffer; this implies a blow from within. Both Teevan and Bergmann have given instances of such lesions; thus, after traversing the cranial cavity, a spent bullet may strike the inner surface of the skull and fracture the overlying outer table alone.

If the force of the blow has been expended by the time the inner table gives way, it alone suffers fracture; if it continues, the outer

gives way as well, but in the latter case it is always to be borne in mind that the inner table splinters over a wider area than the outer. A lesion which is limited to the inner table alone can only occur in a skull well provided with diploë, and consequently in infancy and old age the bone will usually give way throughout its entire thickness at the same moment.

These bending fractures may be associated with little or no displacement of fragments; they may, on the other hand, lead to a marked depression whose floor is made up of firmly wedged fragments from the two tables. They, furthermore, are usually bounded by an irregular circular fissure, into which lines of fracture radiate from the central point of impact. An excellent example of such a circular fracture from bending occurs among the comparatively rare instances of this form of fracture at the base, when, as the result of a fall upon the buttock the impact is transmitted to the occipital bone through the spinal column, and the circular fracture more or less clearly surrounds the foramen magnum.

Distant effects of injuries through bursting.—A diffuse blow from a flat surface is prone to cause effects at a distance, just as a concentrated one from a small body is apt to produce local effects. A bursting fracture of typical form, comparable to the lesions, which Von Bruns has produced experimentally by compressing skulls in a vise to the point of fracture, was cited in the clinical note given above, but it is unusual for the head to be caught and squeezed in this way. An analogous injury may occur when, lying on a hard surface, it is struck by a falling body, though a violent blow against one side of the cranium alone—the head itself usually being the moving force—is the more common method. Though the striking surface, favorable for a bursting effect, should be a flat one, it is common enough for some forms of impact, which produce primarily an indentation, to cause a bursting of the skull as well, in case there is no immediate rebound and if the force exerted be sufficient. Thus, we often find meridional fissures which radiate from a local bending fracture situated at the pole of impact or, indeed, even in the absence of a polar fracture.

Thus, most fissured fractures are an expression of the indirect or bursting effect of a blow, and inasmuch as the base of the skull is more fragile than the vault these fissures occur most readily in this region.

Having oftentimes no apparent connection with any lesion at the point of impact they naturally are spoken of as the indirect result of violence.

The view that these injuries at a distance are due to the effects of a counterinjury or *contrecoup*—a term introduced by French surgeons in the latter half of the eighteenth century—is one which remains popular, though it has been shown to be mechanically wrong.

We learn from these observations that bursting fractures need not be associated with any displacement of bone, but that linear cracks occur which have a tendency to run into the nearest weak portion of the cranial base. These cracks or fissures enter the middle cranial fossæ more often than the anterior or posterior, and it is Walton's view that they often seek out the sella turcica, which presumably is the weakest point of all.

There are factors other than those already mentioned which are thought to modify the direction of these cracks from bursting. Among them are the foramina and the sutures. Whether the fractures tend to seek or to avoid the basal foramina is a matter of dispute. It seems to depend upon the relative strength of the rim of the foramen and the neighboring bone. When the rim is thickened and strong, even so large a defect as the foramen magnum need not be an evidence of local weakness; no more need a trephine opening in the vault in any way weaken the elastic strength of the cranial sphere. Nevertheless, there are certain foramina which are apt to be involved, as the posterior lacerated space, the foramen ovale, and the facial and acoustic foramina.

The sutures, on the other hand, often serve to deflect fissures from the direction which they should have taken by mechanical laws. This is especially true for the skulls of young individuals. (See Fractures by Diastasis.)

The explosive effect of hydrodynamic force.—The introduction of the modern firearm, with its peculiar nondeforming, hard-mantled projectile, has brought an entirely new element into the mechanism of penetrating gunshot wounds, particularly those involving the cranium. It has been made the subject of special study by Kocher, von Bruns, Coler, and Schjerning. When such a projectile, with its extreme initial velocity and great penetrating power, traverses the incompressible semifluid brain, inclosed as it is within a solid covering, it exerts an enormous explosive (hydrodynamic) force against the inner cranial surface. Were the cranial chamber empty a simple penetrating wound of entrance and exit would result, but, being full, the tremendous force is transmitted against all points of its inner surface, and consequently its walls become shattered into fragments.

(b) **Clinical varieties of fracture.**—By the qualification *simple* or *compound* or, possibly better, *open* or *closed*, we indicate, as in skeletal lesions elsewhere, that the fracture is covered by intact soft parts or communicates with the air through an external wound. The distinction is possibly of less vital significance than formerly and, indeed, here more often than in any other part of the body we deliberately, by operative explorations, turn simple fractures into

open ones for the replacement of dislocated fragments or to avoid other complications. In compound fractures of the base, however, we are almost as helpless in the prevention of infection as were our predecessors; for when these injuries communicate with the ear, the pharynx or the sinuses accessory to the nasal cavity, where pathogenic organisms lurk, a doorway which we can not reach is opened to infection. A compound fracture of the base from other cause than bursting is unusual, though it may occur when a weapon, a bullet, or other missile has entered the skull from below. In such a case careful surgical cleansing and drainage is demanded.

Fractures according to their form.—Fissured or linear fractures, as we have seen, are the usual result of bursting; they tend to take a meridional course, radiating from the pole of impact, and, further, owing to its structural weakness, they more commonly occur at the base. When the skull resumes its former shape, after the moment of deformation which causes the bones to spring apart, the fissure will close tightly, provided there has been no associated fragmentation. At the moment of separation of a fissure, substances like hair, portions of headgear, or pieces of the missile which inflicted the blow may either be introduced wholly into the cranial chamber or be caught in a vicelike grip when the edges again snap together. It acts like the “meridional” crack in a child’s hollow rubber ball, which gapes when its poles are compressed.

A fissured fracture may occur as a single linear crack, it may fork or branch, or there may be multiple fissures radiating from the point of impact. A simple linear fissure may close so snugly as to be difficult of detection even on direct exposure. Attention may be called to it, however, by the extrusion, along the closely approximated edges, of fine drops of blood. After death this does not help and at autopsy fissures may escape other than the closest scrutiny. In other instances, whether from interposition of tissue or from some dislocation of fragments, an extensive meridional crack may continue to gape. Such a condition, especially when the fissure has included the vault, may be detected by percussion, or when the head has been shaved, by auscultation combined with percussion, the blow eliciting a “hollow-cask” sound. Furthermore, there will be tenderness along the line of fracture, though this is of little aid in unconscious patients. It is to be remembered that sutures are often mistaken for fissures.

Linear fractures, though simple in themselves, are especially prone to be accompanied by intracranial complications, for their very presence indicates a diffuse blow the effect of which is usually widespread. Hence cerebral contusions are common. The fissures often run across the meningeal grooves and lead to extradural extravasa-

tions and more or less subdural hemorrhage is the rule in the linear fractures of the base.

The treatment, therefore, resolves itself into the treatment of the complications rather than of the fracture, viz, the evacuation of the clot in an extradural hemorrhage; the drainage of the cerebrospinal space if subdural hemorrhage or edema has been sufficient to cause bulbar symptoms.

Fractures by diastasis.—Linear fractures may be deflected into one of the sutures, due to the fact that, before the complete obliteration which they may undergo late in life, they offer less resistance to the cranial deformation than does the bone elsewhere. In the young the lesion may occur as a true separation of the bones. I have seen at operation upon a child of 12, whose head some days before had been caught and laterally squeezed, a simple diastasis of the coronal suture, which had torn the dura, leading to the escape of cerebrospinal fluid under the scalp (spurious meningocele); at no time had there been any cerebral symptoms. In the adult, on the other hand, the process is necessarily more than a simple diastasis, for union is so firm, owing to the close dovetailing of the irregular bony margins, that separation of the sutures can not occur otherwise than by a break.

Comminuted fractures are those characterized by more or less fragmentation or splintering. The comminution may be confined to the area of impact or the entire cranium may be broken into pieces. Being a common result of local deformation or inbending at the point of injury, they are usually situated on the vault and depend for their production on the character not only of the blow (a sharp one with quick rebound) but also of the striking body.

From the standpoint of the bony lesion itself they are more serious than linear fractures, owing to the usual displacement of fragments—**fractures with depression**. Extensive comminution, however, may occur with little, if any, dislocation of the broken pieces. We may, furthermore, in the elastic skulls of infants have depression with no comminution or, indeed, with a total absence of fracture. Finally, in certain rare cases, fracture may occur with actual elevation of a fragment. These effects, however, are unusual; comminution and depression commonly go hand in hand. Hence they will be considered together.

The comminution and depression may affect the inner table alone or both tables, in which case the fragments may consist of the entire thickness of the skull or, in diploëtic skulls, of the separated tables. When thus separated the fragmentation of the inner is always more widespread than that of the outer. The fragments may form a cup-shaped depression, often termed by English writers "pond fracture,"

or they may become tilted at the periphery and slip under the intact cranial edge. We thus have *peripheral* or *central depressions*.

From the pole of impact in comminuted fractures there are often numerous radiating or meridional fissures; these in turn are often connected by zonal lines of fracture, like the connecting strands of a spider's web; and in these cases the farther from the point of impact, the farther apart are the zonal lines, and consequently the larger the fragments. When comminution is the result of diffuse blows, as in the skull of the "butting" negro in the Surgeon General's Museum, or when it follows falls from a great height, irregular fragmentation, like a broken eggshell, may occur, with fissures having no definite configuration.

Almost all punctured or penetrating wounds are accompanied by more or less local fragmentation, with depressions, which particularly affects the inner table.

Depressed fragments may heal in place and their irregularities become, in the course of time, largely smoothed off.

Perforating fractures are due to cuts, to stab wounds, to the penetration of sharp tools which have fallen from a height, to the blow of a pick, the thrust of a bayonet, and what not. They are associated with more or less fissuring, with fragmentation, and with depression of fragments, especially of those broken from the inner table about the margin of the wound. Their course, diagnosis, complications, and treatment do not differ materially from that of wounds of the skull (p. 63), unassociated with fracturing, though produced by similar agencies. When a portion of bone has been carried away, leaving a defect, they are called *fractures with loss of substance*.

One particular group of perforating fractures deserves special consideration; namely, those which are the result of wounds from fire-arms.

Gunshot fractures.—In their simplest form these are perforating fractures which produce a circular loss of substance. When the result of a wound at short range from the modern small arm, we have seen that the skull may be burst outward by the explosive action of hydrodynamic pressure. We have learned, too, that these are compound or open fractures; that they are almost always comminuted ones with some depression of fragments; and, finally, that they often lead to sepsis, hemorrhage, or other intracranial lesions, which make of them a particularly dangerous and crippling form of injury.

There are, however, other types of gunshot fracture less serious, since they are nonperforating. Thus, the direct impact of a heavy spent ball may fissure or indent the skull without producing more than a bruise of the scalp. Again, a bullet may pass through the scalp and graze the vault in a tangential direction without penetration, or it may furrow the bone, scooping out a gutter in the outer

table alone or leaving a defect of the entire cranial thickness. The lateral force exerted, during its rapid flight, by the modern high-velocity projectile is sufficient to comminute the skull, even if it be merely grazed, so that only in those localities where the bone is thick and porous or contain air cells would it be likely to escape considerable local fragmentation from such a tangential wound. For the same reason fractures of the base may occur when a bullet traverses the shell-like bones comprising the under surface of the skull without actually penetrating the cranial chamber; and, further, bullets may become lodged in the thicker parts of the cranial wall and produce more or less local comminution without actually entering the cavity.

The wound of the bone, as we have seen, may show nothing more than a clean-cut circular or oval loss of substance, but it is the rule for the wounds both of entrance and exit, in case they occur in bones containing diploë, to show the particular characteristics of punctured fractures; that is, to have a more or less splintered margin, particularly of the table which has been last penetrated—the inner table for the wound of entrance, the outer table for that of exit. Hence, when there is a loss of substance due to the actual carrying away of fragments, the circumference of the defect will be greater on the side from which the missile has emerged, whether it be wound of entrance or exit—a matter often of medicolegal importance. Furthermore, meridional fissures are apt to radiate from the wounds of impact, and these meridians in turn are often joined by circular fissures on zonal planes.

The damage from perforating bullets depends partly upon the physical properties of the missile and partly upon the speed with which it is traveling. There is great difference, therefore, between the effects of the soft, leaden bullet discharged from a revolver and that of the modern conical projectile with its hard mantle and tremendous initial velocity. The latter missiles, except near the end of their flight, rarely lodge; the former almost invariably do.

In his *Handbuch der Praktischen Chirurgie*, Bergmann gave in full the results of experiments conducted by himself and others upon wounds of the head made by the modern rifle. Briefly, it may be said that at close range the skull and scalp are literally torn to pieces and the brain disorganized; that on penetration at 50 yards the scalp remains intact, though the skull is greatly comminuted and brain tissue oozes both from the wound of entrance and exit; at 100 yards there occur zonal fractures which tend to be limited to the area about the wounds of entrance and exit, while meridional fissures radiate from these points, showing that explosive action is still effective; at 1,000 yards the zonal cracks encircling the bullet holes disappear, and only the radial fissures remain; at the distance of 1 mile the fissures largely disappear, leaving the two clean-cut bullet holes; and not until over $1\frac{1}{2}$ miles does the projectile fail to emerge after entering the skull on one side. All this, of course, is merely relative, for there would be great difference, not only in individual skulls, but

in the position in which they were struck; and it is, after all, a matter chiefly of interest to the military surgeon.

On the whole, these wounds in warfare have a grave prognosis. According to Fischer's statistics from the German Army during the Franco-Prussian War, 45 per cent of 8,132 gunshot injuries resulted in immediate death, and nearly one-half of those found dead on the field of battle had wounds of the skull.

The gunshot fractures which are seen in civil life are more apt to result from revolver shots and in orderly communities to be self-inflicted wounds, whether by accident or intent. Here again the character of the injury depends upon the nature of the weapon and the initial velocity of the projectile. Most of the wounds which one sees to-day are produced by soft bullets fired from the ordinary revolver with no great initial momentum; the heavier army pistols, on the other hand, fire a projectile which at near range has the explosive effect of a rifle. The soft, deforming revolver bullets are apt to lodge either in the bone at the site of entrance or somewhere within the cranial cavity, either at some point in the direct line of their flight or, in case the missile has rebounded, at some point in a line determined by the angle of deflection from the opposite inner surface. At times the course of such a deflected bullet may be mathematically calculated, but to-day the *x*-rays give us a more accurate means for determining its position. It may be said, however, that, as a rule, there is no particular reason for its extraction, for in the absence of immediate complications it becomes encapsulated and, unless the missile chance to lie near the surface, the damage already done will only be increased by meddlesome attempts to locate and extract it.

The complications which result from penetrating bullet wounds may be classified as *immediate*, or those due to hemorrhage, compression, and destruction of tracts; *intermediate*, or those due to sepsis; and *late symptoms* (irritative and paralytic), giving evidence of the permanent damage done to the cerebral tissues. Hemorrhage, of all the immediate symptoms, is especially to be dreaded, as it may lead to rapid death from compression. In all cases in which there is an increase in intracranial tension from effused blood, the pressure forces the disorganized nervous tissue through the wounds of entrance and exit, and the extruded particles of white tissue are found mingled with the blood which oozes from the opening. It is not uncommon for cranial nerves to be injured, either by direct section or by implication in a basal fracture.

It is notorious that suicides often fail to accomplish their purpose. Brun has recorded 32 cases, of which number 16 recovered. Of these cases the wound of entrance was in the right temporal region sixteen times, in the left twice, in the forehead nine times, and in the mouth twice. The "temple" is regarded by the laity as a particularly vulnerable spot, which accounts for the preponderance of the attempts in this situation. In them oftentimes the bullet merely

passes extra-cranially from temporal fossa to temporal fossa, through the back of both orbits, cutting the optic nerves and leading to blindness—a sad penalty for a criminal act. Though unrecorded in Brun's series, suicidal wounds inflicted in the right mastoid region are not uncommon.

The later complications, in case of "recovery," are paralyses, mental changes, epilepsy, etc. Thus, a patient was accidentally shot in the mid-line of the forehead at the hair margin. A surgeon removed two pieces of the bullet (supposedly all of it), together with some fragments of bone at the wound of entrance, which finally healed. The patient for a time was aphasic, had a left-sided hemiplegia, and he subsequently developed epilepsy with a peculiar speech aura. An *x*-ray plate then showed a foreign body lodged in the left side of the brain near the skull, and just below the middle of the Sylvian fissure. An operation was performed, adhesions due to an absorbed subdural clot were separated, and a small, dense scar, inclosing the main fragment of the bullet, was removed. This had traversed the left frontal lobe, had struck the side of the skull, and ricocheted into its position. The extraction of the bullet benefited him in no respect, and he is progressing to mental degeneracy.

Treatment.—The fracture itself is the least of the ills following cranial gunshot wounds and can not be considered apart from the other complications. If there is a clean-cut perforation and no serious immediate symptoms the wound may be left with a simple drain and healing may take place without incident; for unless septic foreign particles have been carried in with the missile, its track quickly cicatrizes and the bullet itself becomes encapsulated. If there is a lacerated scalp and considerable local comminution of the skull it is advisable, after paring the edges of the scalp wound, to enlarge it by incision and to trephine the skull in order to readjust any depressed fragments, to evacuate clots, to relieve tension, and to afford better drainage. A large defect almost always leads to a hernia and perhaps to a fungus cerebri, owing to the swelling of the lacerated brain. Largely owing to this, drainage of the track of the bullet is a most unsatisfactory procedure, and one must usually be satisfied with a superficial drain down to the dura and brain, but not far into the latter. The temptation to probe for, to locate, and to extract deep-lying fragments of the bullet should be resisted by the surgeon; for even if successful in their object these procedures usually serve merely to increase the damage already done by the missile without conferring any benefit whatever from its removal.

The late complications must be met as are those due to cranial injuries from other causes, and here again it must be borne in mind that the paralyses and mental disturbances are not due to the presence

of the foreign body, but to the cicatricial changes in the nervous tissue due to its passage through them, and that they consequently are the same whether the bullet has lodged, emerged, or been removed.

Fractures according to their situation—Fractures of the vault and fractures of the base.—There is a certain justification in this common anatomical division, for in the two situations not only do fractures differ in the mechanism of their production but also in the form which they assume and in the complications to which they are liable. The bones of the exposed vault are more liable to direct, indenting injuries, and hence, despite their greater strength, comminution with dislocation of fragments is frequent; those of the well-protected, though more fragile base, are more subject to fissuring, the result of general deformation of the skull. These, of course, are not invariable rules, for we may have simple fissures of the vault from bursting or a local comminution with depression at the base from bending—an example of which is the not uncommon fracture which occurs about the foramen magnum as the result of falls on the buttock when a direct blow is transmitted to the base through the spinal column. Again, a simple bending fracture of the base may follow a sharp blow on the chin, when the ramus and condyle of the jaw transmit the force to the base—the prize-fighter's fracture. Finally, it is always to be kept in mind that fractures of vault and of base are apt to be associated.

Many statistical studies in regard to cranial fractures have been made from time to time; notable among them is the recent elaborate monograph of Hans Brun, based on 470 cases which in 20 years had been carefully observed in the Cantonal Hospital in Zurich.

Occurrence.—In general it may be said that they are injuries of young adult life; that they are many times more frequent in men than in women; that in the majority of cases (60 per cent) they are the result of falls from a height. About one-half of these fractures involve the base, often alone, sometimes with associated fracturing of the vault. On the other hand, it is estimated that 70 to 75 per cent of all fractures of the vault are accompanied by basal injuries. Fractures of the vault are more often compound than simple, and they occur with about equal frequency in frontal, parietal, and temporal bones, being rare in the occipital region. Basal fractures are more common in the midcranial fossæ.

Mortality.—Disregarding the etiological factor, the patient's age, and also the character of the injury, about one-third of all cases in the past have proved fatal, and as the fatalities are largely due to the immediate cerebral complications, modern methods of treatment have not served to greatly alter these figures. The percentage of fatalities increases with age—the younger the individual the more favorable

the outcome. Fractures of the base are commonly thought to be attended with a higher mortality than those of the vault, though with our improved diagnostic measures (lumbar puncture, for example) we may find that many cases of simple basal fracture have heretofore been overlooked and regarded merely as concussion—a fact which may make one's percentage of recoveries at least appear larger to-day. Excluding those cases which have died as an immediate result of the injury and those which have later succumbed to infection, the average duration of life in the fatal cases is said to be 44 hours; so that there is some basis for the old rule, adhered to by Bergmann and Wagner, that survival over two days gives a favorable prognosis.

Of the cases which survive the first 48 hours, a considerable number (8 per cent of all fatalities) die from the intermediate complications of meningitis or abscess. Fractures of the base are more liable to this complication than those of the vault; for the latter are accessible and easily drained, so that, unless there be a defect leading to a fungus cerebri, dangerous from a persisting leak of cerebrospinal fluid, infection rarely occurs. Basal fractures, on the other hand, especially those which open up the sphenoidal or ethmoidal sinuses where pathogenic organisms lurk, are often followed by a meningeal infection. In this case the pneumococcus is the more common agent, whereas in fractures of the vault a streptococcal or staphylococcal infection is the usual one. I have twice seen a rapidly fatal pneumococcal meningitis start up on the third day after what appeared to be a simple, uncomplicated basal fracture with a little bleeding from the nose and so few subjective symptoms that the patients remonstrated at their enforced recumbency.

The *prognosis* is in no way proportionate to the extent of the cranial injury, but depends entirely on the character of the intracranial lesions which will be fully considered anon. An insignificant crack of the base, associated with a focal hemorrhage in pons or medulla, may put a sudden end to life; whereas an extensive fragmentation of the vault, which allows for considerable cerebral expansion, may actually save life through "decompression." An insignificant punctured fracture which does not even produce concussion may prove fatal from meningitis or abscess later on; a comminuted and depressed compound fracture may, on the one hand, cause death quickly from hemorrhage and compression, or may heal practically untreated and give few symptoms. On the whole the immediate prognosis is more favorable in bending fractures than in bursting fractures; in other words, more favorable in those which are accessible than in those which are not, for it depends largely on the possibility of early treatment not only of the fracture, but of its underlying complications.

The *diagnosis* of fractures of the vault may offer difficulties, particularly in the case of linear fissures and of those involving the inner table alone. One, however, is much more apt to be misled by the peculiar feel of the infiltrated edge of a subaponeurotic extravasation into making a faulty diagnosis than to overlook a cranial fracture when it is actually present. When the scalp is intact, linear fractures may at times be recognized through lines of tenderness on pressure, particularly over the temporal fossa, and by a changed percussion note if there is any gaping of the fissure. In open wounds there should be no difficulty in recognizing even a closely approximated fissure, owing to the blood which oozes from between its edges; sutures, however, may be mistaken for fissures. Localizing cerebral symptoms, to be discussed later, are often helpful in determining the situation of a depression if it is not actually palpable, and involvement of cerebral nerves may indicate the direction taken by a meridional fissure. Old deformities dating from birth, patches of senile atrophy, defects from former diseases, like syphilitic osteomyelitis, and the irregularities present in all skulls, though more marked in some, may at times be mistaken for depression.

In fractures involving the base alone we must, in the long run, depend entirely upon the symptoms which we have learned to recognize as common accompaniments of these injuries, rather than upon any direct evidence of the bony lesion. Evidence from intracranial or extracranial bleeding, either free or into the tissues, is of particular value.

The intracranial extravasations usually take place into the subdural space, for, owing to its close attachment, the dura is usually torn when the bones are fissured. The amount may be small or so extensive as to cause rapidly fatal compression. It may be recognized by finding evenly distributed red blood corpuscles in the cerebrospinal fluid withdrawn from the lumbar meninges.

The extracranial extravasations may also be free and bleeding may occur from the nose, mouth, or ears, in case the ethmoid, the accessory sinuses, the Eustachian tube, or the tympanic cavity have been implicated. It is necessary to exclude a simple "bloody nose," rupture of the tympanum, or entry of blood from without into the auditory canal. Extravasations into the tissues (ecchymoses) appear more tardily. They are common in the orbit, under the eyelids or conjunctiva when the frontal plate is injured, and in fractures of the middle or posterior fossæ they find their way to the surface over the mastoid process or down the neck after some days.

The escape of cerebrospinal fluid often occurs with fractures entering the middle fossa, particularly when they involve the petrous bone and when both dura and tympanum have been torn. The escape

of bloody fluid may continue for days and the symptom need not always be entirely undesirable, as pressure may be relieved thereby. Open fractures which communicate with the nasal or pharyngeal cavities may likewise be followed by a leakage of cerebrospinal fluid, though it is less common from these situations. In rare cases, after a lesion of the petrous bone unaccompanied by rupture of the tympanic membrane, fluid may escape into the pharynx by way of the Eustachian tube and either be swallowed or flow from one nostril when the head is tilted down.

The *complications* of cranial fractures often serve as an aid in diagnosis. They are estimated to occur in 46 per cent of fractures of the vault and in 64 per cent of those of the base. Varying grades of concussion, contusion, or compression are almost inevitable; only exceptional forms of fracture occur without one or another of these classical symptoms, though any one of them may result from an injury in the absence of fracture. They are apt to be more outspoken in basal lesions, owing to the diffuse character of the blow necessary to produce a bursting fracture. As will be described in its proper section, compression may be general or local, and when local it may give cerebral symptoms of irritation or of paralysis, which serve to point out the situation and character of the cranial lesion. This is often the case with indented fractures of the vault which lead to cortical laceration of the brain, or with meridional fissures which cross the meningeal groove and lead to extradural hemorrhages with their characteristic "interval" between symptoms. These extradural hemorrhages can only occur under the vault where the dura is more easily separable from the bone than at the base; and it is to be remembered that they are not necessarily an indication of fracture, but may be the result of simple deformation not sufficient to break an elastic skull.

Involvement of the *cerebral nerves* may prove a valuable diagnostic aid. The facial is by far the most commonly injured, owing to its devious course through the petrous process, so frequently implicated in fissures entering the middle fossa. The mere presence of hemifacial palsy, however, after an injury to the head need not indicate with certainty the peripheral involvement of the nerve, for it may be due to a contralateral central lesion. In order of frequency the abducens comes after the facial, and diplopia from an involvement of any or all of the oculomotor nerves may be the result of breaks in the neighborhood of the sphenoidal fissure. In fissures crossing the frontal fossæ the olfactory often suffers. The optic may be affected by direct injury, and lesions of the trigeminus, glossopharyngeal, vagus, spinal accessory, and hypoglossal have been recorded. The nerves are apt to be affected in groups, the seventh, eighth, and sixth together; the fifth and third; or the ninth, tenth, and eleventh. The

lesions usually occur at or near their foramina of exit, due either to actual laceration or to local compression from effused blood; and hence a study of the paralyses may indicate the fossa which the fracture has entered, whether anterior, middle, or posterior.

Serious complications from hemorrhage may follow *injuries to blood vessels*, particularly when the sinuses or meningeal artery are lacerated, and occasionally linear fractures crossing the middle fossa toward the pituitary fossa may so traumatize the cavernous sinus and carotid as to produce an arteriovenous aneurysm with pulsating exophthalmos.

The sequels heretofore considered are common to all lesions of the skull; those due to an *infection* are almost without exception limited to open fractures, whether of base or vault. In them purulent cellulitis, osteomyelitis, septic sinus thrombosis, meningitis, or cerebral abscess were formerly almost to be expected. Modern methods have largely lessened these evils in the case of the vault, even if not in basal lesions.

An insignificant fissure which passes across the ethmoid plate may open a pathway of infection from the nasal cavity and lead to a rapidly fatal meningitis. Cerebral abscess is especially common after punctured or gunshot wounds from the deep inoculation of infective agents, though it occurs often enough in compound comminuted fractures which have led merely to a superficial laceration of the cortex.

Rarer complications, like spurious meningocele, pneumatocele, and others too numerous to mention, may likewise occur. Cysts occasionally form after fractures either from a torn dura, from the partial absorption of a clot in the subdural space, or from a subcortical extravasation. Sugar may appear in the urine (traumatic glycosuria), usually about 8 to 12 hours after the injury and in about 9 per cent of all cases, according to Higgins and Ogden. All of these, as well as the so-called post-traumatic neuroses, result from the cerebral, not from the cranial injury.

The *process of healing* does not take place as in the long bones, where there is an abundant callus formation. Dura and periosteum, however, are both capable of forming new bone, as we have seen in exostoses, osteophytes, etc., and complete repair, even when there has been a loss of substance, may occur. It may, however, be long delayed or completely fail, due, according to Bergmann, to the destruction or less of the osteoplastic layer of both inner and outer periosteum as well as to the absence of movement which ordinarily stimulates callus formation.

Union often occurs by fibrous membrane alone; even narrow fissures may fail to become reunited by bone. As a rule, however, a slow process of bone production and bone absorption goes on, hand in

hand, and irregular edges or depressed fragments are rounded off as the gaps are more or less filled in. Occasionally there is an overproduction of new bone, leading to focal or to widespread hyperostoses from either the outer or inner table. Even defects of some size may at times become entirely ossified, and even when closed by membrane alone they may become so firm and inelastic as to show no pulsation. According to Bergmann, defects can not be expected to close if they exceed a diameter of 6 to 8 centimeters, and it would indeed seem that it is rare even for much smaller openings to fill in.

There is a great difference of opinion as to the injurious effect of these bone defects, some holding the view that when extensive they lead, in the course of time, to serious mental symptoms. Personally I do not believe that they are injurious unless accompanied by an underlying lesion of the dura. When the dura is wounded and the scar formation leads to adhesions between overlying scalp and brain, the chronic fibrous changes which result may lead in time to extensive cortical alterations and mental deterioration. With an intact dura, however, such symptoms are less likely to occur.

Treatment.—We are confronted again by the necessity of distinguishing between the management of the fracture itself and the management of its complications. Relatively, simple rules can be laid down for the former; for the latter our conduct is largely controlled by physiological laws relating to the circulation of the blood and cerebrospinal fluid under abnormal conditions. In fractures of the vault the indication for surgical intervention is usually deformation of fragments, rather than critical cerebral complications; in fractures of the base it is the reverse, for there intracranial complications are especially serious and deformation is rare.

In compound injuries of the vault we may easily determine the form and estimate the consequences of the injury, and our endeavor should be to thoroughly cleanse the wound, to elevate depressed fragments, to restore a wound in the dura if one exists, and to leave the parts as nearly in their natural position as possible. If the fragments are depressed and wedged it may be necessary to trephine at the edge of the depression before they can be pried into place. Even in the absence of visible depression an opening may be required when cerebral symptoms are present, due to depression from the inner table alone or to intracranial hemorrhage.

It is another matter when injuries of the vault are covered by intact scalp, for there may often be great difficulty in determining whether there is sufficient justification to transform a simple into a compound fracture, even for the sake of determining the lesion. It is largely a personal matter and rests with the judgment of the operator; and this in turn depends entirely upon his familiarity with intracranial disturbances which are amenable to operative treatment

and his ability to safely cope with them when found. A simple fissure, which crosses the temporal region, of itself needs no surgical intervention, but this is urgently called for when pressure symptoms indicate either a lesion of the meningeal or free extravasation at the base. When simple fractures are accompanied by evident depression surgical measures are indicated, even in the absence of immediate cerebral symptoms, for unelevated fragments are almost certain to be the source of future trouble, especially if the dura has been injured. The opening must be carefully examined on all sides, for depressed fragments are readily overlooked. In Fig. 50 is shown a patient with a depressed fracture which had solidly healed, in so far as the union of the displaced fragments was concerned, and the rounding off of their sharp edges, and yet serious mental (left frontal lobe) symptoms resulted. In an old healed depression of this sort it is necessary to remove the entire area by a circular incision and either to leave a defect, to replace it by some foreign material, or to cover it by an osteoplastic flap. Occasionally the cup-shaped area may be replaced inverted without subsequent necrosis.

When there is extensive comminution with many loose fragments it may be difficult to determine whether any of them should be removed, owing to possible loss of viability. The dread of necrosis and of infection, though a natural one, is largely an inheritance from our surgical forefathers, and it is a matter of present-day experience to find that fragments of surprising size, even when completely separated, will survive if left in a clean wound. It formerly was the custom to remove all completely detached pieces. It has been learned, however, that even boiled fragments may heal after reinsertion. I have quite frequently replaced a 3-cm. trephine button after boiling it (owing to some suspicion of its perfect cleanliness), and have never seen it fail to heal in place; it is well known, of course, that an unboiled fragment of this size can always be safely replaced. In the latter case it is probable that bone-forming cells of either surface or of the diploë may remain viable, but in the former instance they have been destroyed and it is to be presumed that the fragment acts only as a temporary stimulus for new tissue, becoming itself ultimately absorbed. The question of closure of defects will be considered later.

The treatment of basal fractures resolves itself largely into the treatment of contusion or compression of varying degrees, for which our therapy is largely restricted to rest, absolute quiet, an ice cap, sedatives when headache is severe or when there is great restlessness, and to free evacuation of the bowels, preferably with a saline—measures to be observed in practically all cases of cranial injury. The greatest care should always be exercised in handling and in trans-

porting any case of fracture with intracranial symptoms, for the symptoms are much aggravated by any form of jolting.

With the view of preventing infection in case there is bleeding or loss of cerebrospinal fluid from the nose or ears, it is customary to irrigate and tamponade the auditory meatus or the nasal chamber. This procedure, however, can be overdone, and only in case the hemorrhage is profuse is it justifiable to actually pack these orifices, for nothing is more certain to set up a suppurative infection of the mucous membrane. Irrigation of the nose or of the ear to remove clotted blood and to cleanse the cavities has an element of danger, and it is preferable to accomplish this by merely wiping out the passages with a sterile cotton swab moistened in a mild antiseptic solution. It is important not to irrigate the Schneiderian membrane in such a way as to produce a sneeze, for on more than one occasion this has been disastrous and the explosive effect has driven septic material into the middle ear or ethmoidal cells and apparently has been the active agent in inaugurating a septic meningitis. After cleansing, the cavities should be loosely closed by a wisp of absorbent cotton, or of iodoform gauze if desired, which will serve to take up the secretions and which should be frequently changed if there is abundant discharge. Although by energetic measures we may overcome a local meningeal infection which has started over the hemisphere, we stand practically helpless before one originating at the base, although suboccipital drainage in a few cases has apparently resulted in cure.

One can speak somewhat more encouragingly in regard to active interference in case of diffuse hemorrhage. The fatalities from this cause, as will be detailed in the section dealing with compression, are due to a final implication of the vital centers in the medulla when the amount of effused blood is sufficient to so increase intracranial tension that they are thrown out of function from anemia. Though this has been well recognized, operative methods of meeting the situation have been inefficient or untried, owing to the feeling of hopelessness in the presence of continual oozing from an inaccessible and often uncertain lesion. Exploratory openings have usually been made over the vault, but, owing to the increased cerebral tension, such openings become filled with a bulging brain, drainage can not be effective, and a fungus cerebri is often produced.

It is self-evident that an opening as near the lesion as possible is desirable, and, inasmuch as most of these fractures enter the middle fossa, an opening low down in the temporal region is most likely to be efficacious. The author's procedure, which has been designated as an intermusculotemporal operation, often meets the needs of the condition. In this operation, by splitting the temporal

muscle in line of its fibers and by rongeur-ing away the thin squamous wing of temporal and adjoining sphenoid, not only is the region overlying the meningeal vessels exposed, so that a chance extradural hemorrhage can be brought to light, but also the dura over the temporal lobes is exposed and the presence or absence of subdural effusion can be determined. The dura should be opened and there will usually be an abundant escape of bloody cerebrospinal fluid, whose evacuation will be aided by passing a curved, blunt dissector down under the temporal lobe. Oftentimes edema, as we have seen, has played the chief rôle in the compression, and we may find that merely a so-called "serous meningitis" is present, and that evacuation of a large amount of fluid will diminish the tension. In such case the muscle and scalp may be closed, but if there is continuous bleeding it is well to close the muscle only in part and to leave, at its lower angle, a strip of rubber protective, leading under the temporal lobe as a drain. In case the craniectomy on one side alone seems insufficient a bilateral operation may be performed at the same or at a subsequent sitting, for the procedure is simple and not attended by shock. Its advantages are due to (1) the frequency of the bony lesion in the middle fossa; (2) the fact that cerebral contusions are especially liable to involve the tip of the temporal lobe; (3) the exposure of the meningeal territory and ease of determining the presence of an extradural hemorrhage; (4) the possibility of draining through a split muscle rather than directly through the scalp; and (5) the subsequent protective action of the muscle in case a hernia tends to form in consequence of traumatic edema. The unilateral or bilateral defect in this situation leads to no complications and no subsequent deformity.

PART 2.

MENINGES, EPENDYMA, AND BRAIN.

MENINGES AND EPENDYMA.

Anatomic and physiologic considerations—The pachymeninx and its vessels.—A thorough knowledge of these membranes and the part played in intracranial disease by the fluids which they hold is of prime importance. It is permissible to call attention here to some of the more essential points only.

The *dura* carries on its outer surface certain arteries of surgical interest and it incloses the great venous sinuses. The intracranial *dura* differs from the intraspinal *dura* in its relation to the inclosing bone; for the latter has a double, the former only a single inner layer of endothelial cells, its outer surface adhering more or less firmly to the skull and having, especially in the young, an active share in the process of bone formation. This attachment is an intimate one, particularly at the base, from portions of which the *dura* can be separated only with great difficulty. Hence in linear fractures of the base the membrane is almost always torn; in similar fractures of the vault it may often escape injury. One source of its firm adherence is to be found in the sheath-like prolongation of the membrane along the course of the cerebral nerves. Such a sheath is especially marked in the case of the optic nerve—a fact of importance in the etiology of choked disk. Owing to this firm basal attachment extradural hemorrhages are less likely to occur there than under the vault; this is not an invariable rule, for in certain parts of the middle and posterior fossæ it is readily separable from the bone—a fact which is made use of in exposing the trigeminal nerve and in suboccipital operations. The strength of the dural attachment at the vault is variable; it increases with age, so that the membrane tears in removal; in the young it clings much more firmly to the growing bone at the sutures than elsewhere. In the newborn, for this reason, an extradural hemorrhage ("*internal cephalhematoma*") may be limited to the inner surface of one bone.

As a protection for the brain, the *dura*, owing to its smooth endothelial surface, is of much more importance than the overlying bone. A dural defect is replaced by scar tissue, which of necessity leads to adhesions between the cortical leptomeninges and overlying cranium

or scalp, as the case may be; a dural wound, the edges of which have been accurately approximated, should leave no such adhesions, as the edge is quickly united by proliferation of the endothelial cells. The dura is, in a measure, separable by dissection into two layers—an outer and inner—between which structures like the Gasserian ganglion are inclosed; it furthermore opens to inclose the large sinuses, from the inner edge of which falx and tentorium pass off to partition the cranial space into its three main chambers. These strong membranous partitions play an important part in supporting the hemispheres, and, inasmuch as they can be dislocated but slightly out of their normal position, they have a tendency to limit the pressure effects of a local process to the one compartment in which it has originated. This is especially so with the subtentorial compartment, for the tent like membrane hung from the posterior edge of the falx is particularly well adapted to support pressure from above and thus protect the important centers of pons and medulla.

The dura deserves chief consideration as a carrier of blood vessels. The superior *longitudinal sinus* lies slightly to the right of the median line. It increases in its blood-holding capacity as it runs from the ethmoid to the torcular. In its course it changes greatly in form. On cross-section, except during its middle course, it is of a narrow, wedge shape, the apex of the wedge running down for a considerable distance between the two layers of the falx. During its middle course broad expansions (*lacunæ laterales* or *parasinoidal sinuses*) pass out between the two dural layers for a distance of from 1.5 to 2.5 cm. over the hemisphere. Into these lateral expansions enter many of the more important of the superior cerebral veins, particularly those which ascend in the sulci bounding the paracentral convolutions, and from them emissary vessels pass through the inner table to communicate with the diploëtic vessels.

Into the *lacunæ laterales* project the majority of those bodies known as *Pacchionian granulations*. They seem to be an acquirement of adult life and their function, if they possess any, is uncertain. They consist of tuftlike processes from the arachnoid containing cerebrospinal fluid; and, covered merely by a thinned-out layer of dural endothelium, they project into and are bathed in the blood of the sinus. As they enlarge they may even project through the lateral expansion of the sinus and cause, through pressure, atrophic depressions of varying size in the under surface of the skull. It is for these reasons that injuries to the skull in this situation or efforts to operate there are especially likely to be attended by hemorrhage; for the calvarium cannot be separated from the dura without tearing the emissary veins or injuring the thinned-out layers of the dura covering the granulations. Furthermore, the dura itself can not be elevated from the brain without injury to the cerebral

veins, which, as well as the Pacchionian granulations, serve to bind it to the cortex. Since the *lacunæ laterales* are sufficiently broad to cover the motor centers for the lower extremities, it is evident that these centers are difficult of access. Owing to the *lacunæ* also, ligation of this sinus in its middle course is almost impossible. The longitudinal sinus and its expansions, furthermore, are honeycombed by fibrous bands (*chordæ Willisii*), which pass in various directions and many of which seem to serve as valves at the point of entry for the superior cerebral veins. These vessels enter the sinus by long, obliquely placed channels which pass forward against the direction of the blood current.

At the torcular the longitudinal sinus bifurcates into the *lateral sinuses*, the right being usually the larger. These sinuses lie in the triangular space at the tentorial attachment and, usually with a slight upward convexity, pass to the mastoid region of the skull, where, as the *sigmoid sinus*, with a sharp curve they turn downward to become the internal jugular vein at the foramen of the same name. At the posterior part of the mastoid process there is a foramen for a large emissary vessel which forms a communication between veins of the scalp and the lateral sinus. Injury of these communicating veins in attempting to remove the overlying bone for operative purposes may lead, especially when there is any venous stasis, to profuse hemorrhage, for such an accident amounts to a lateral injury to the sinus itself. Communicating vessels also pass from the sinus to the bone at the occipital protuberance opposite the torcular, and here again especial care must be exercised in removal of bone. During the middle course of the lateral sinuses there are no important vascular communications, and in operations the bone may be rongueured away from the dura without risk of bleeding. Owing to the particularly close attachment of the dura to the petrous bone the petrosal sinuses are important, as they are apt to be injured in the linear fractures which so commonly invade this fragile part of the base. The hemorrhage therefrom either directly enters the subdural space or appears from the external meatus.

A sagittally placed *occipital sinus* connecting torcular with the venous sinuses of the spinal canal lies at the junction of the dura of the posterior fossa and the cerebellar falx. Unlike the falx cerebri this membranous expansion is of variable size and not infrequently both it and its marginal vessel are small or absent. It is my impression that when the sinus is absent its place is taken by large median diploëtic vessels which run in the midoccipital ridge to the edge of the foramen magnum and thence to the condylar foramina. Diploëtic vessels here may prove troublesome in the suboccipital approach to cerebellar operations.

Furthermore, at the base lies another important sinus—the *cavernous*. As it lies alongside of the sella turcica it may be injured by bursting fractures whose fissures so often enter this region, and its companion vessel, the internal carotid artery, may be harmed at the same time, leading to an arteriovenous aneurysm. As it drains the venous blood from the brow and orbit it is liable to infection spreading from these vessels; and when compressed, as by tumor, the resulting venous stasis in the bulbar circulation is supposed by some to be the causal factor in producing a choked disk. When thrombosed it produces an extraordinary degree of exophthalmos, usually resulting in blindness.

There has been much discussion among experimentalists concerning the compressibility of the sinuses, and from their peculiar form and protected situation they have been regarded as incompressible. I succeeded in demonstrating that in the dog the longitudinal sinus may completely collapse at an early stage of compression, with consequent venous stasis of high degree. If, with increased tension from any source, a similar collapse may be produced in the *sinus rectus*, with stasis in the *venæ Galenæ*, an internal hydrocephalus may be produced without the direct implication of these vessels by pressure from a neighboring growth.

The *middle meningeal artery* serves as the chief arterial supply for the dura. Its intimate connection with the membrane is such that in separating dura from skull it clings to the former, though there are often small branches which enter the bone. However, at one place—the anterior inferior angle of the parietal, near the pterion—it grooves or actually may channel the bone, so that separation is often impossible without troublesome hemorrhage. Linear fractures crossing the pterion are also almost certain to injure the vessel.

The nerve supply of the dura is abundant. Unlike the leptomeninges, it contains sensory and vasomotor nerves, the chief supply coming from the trigeminus, although about the foramen magnum there are fibers from the vagus. Headaches I believe to be due to the stretching of the dura or of its expansions.

The leptomeninges, ependyma, and cerebrospinal fluid.—The *pia*, a delicate and vascular membrane, closely hugs the convoluted surface of the central nervous system; fitting like a glove it dips down into all of the fissures and irregularities. The *arachnoid*, on the other hand, bridges over most of the crevices, and in its relation to the pia may be likened to a mitten drawn over the pial glove. On the top of the convolution where these membranes come in contact they are intricately associated; over the sulci and fissures, and particularly over the irregularities of the base, they are more or less widely separated. Unlike the free subdural space, these subarachnoid spaces are honey-

combed by delicate tissue strands which bind pia and arachnoid loosely together. This loose tissue meshwork is traversed by many of the cortical vessels whose tissue support consequently is slight. The cerebrospinal fluid circulates in these spaces.

The *ependyma*, which lines the ventricular cavities, consists of a layer of epithelial cells, underlain, for the most part, by a thin layer of neuroglia. It forms a covering for the velum interpositum and the vascular choroid plexuses which curve backward through the foramina of Monro and project into the posterior cornua of the ventricles. Though morphologists have shown us how these important structures develop, their physiological signification remains obscure, and consequently their diseases even more so. They doubtless play the chief rôle in the formation of cerebrospinal fluid, and it is essential that we have, as a working basis, some knowledge of the function of this fluid.

The *cerebrospinal fluid* is not, as was long conjectured, merely a surface lubricant akin to the fluid of the great serous cavities; nor does it act alone as a water-bed, though the comparatively large collections of fluid in the subtentorial cisterns serve in a measure as a support for the important centers of the hind-brain and lessen the jar which they would otherwise receive in case of a cranial injury. Leonard Hill expressed the opinion that the cerebrospinal fluid should be regarded as the lymph of the brain, but, as Halliburton has shown, it is a true secretion and not an exudation like lymph, from which it has marked chemical differences. The subarachnoid space is only remotely connected with the lymphatic system, being fully developed long before the lymphatics have budded out from the subclavian and iliac veins (Sabin). Observations undertaken for me by Lewis Reford in Mall's laboratory show that the spinal arachnoid is completed first, and in later weeks spreads slowly over the hemispheres. Reford's observations, furthermore, have shown that from the beginning the spinal subarachnoid space is intimately connected with the venous circulation through the rudimentary cerebral sinuses.

There appears to be an active secretion and circulation of this fluid, which, under certain circumstances at least, may form in large amounts. This is shown not only by the copious discharge after certain cases of cranial or spinal injury or in nasal rhinorrhea, but also by the rapid reaccumulation of fluid in a hydrocephalic head after its withdrawal.

Of the manner in which this fluid gathers we are as yet unaware. It may be entirely a transudation from the capillaries of the vascular choroid—a view held by Leonard Hill—or, what seems to me more probable, it may be largely the product of secretory activity of the

ependymal cells which line the choroid plexus, the function of which has not been definitely established. However this may be, the fluid originates in the lateral ventricles, whence, passing backward by the median ventricle to the hind-brain, it escapes into the subarachnoid space by way of the so-called foramina of Magendie and of Luschka. From this point the fluid bathes both the cord and brain. It is chiefly, though not entirely, confined to the ventricular and subarachnoid spaces. Outside of the arachnoid there normally exists little free fluid, though it may gradually make its way through this membrane. When exposed in an operation the arachnoid may be seen to exude drops of fluid—to sweat, as it were—but not until it has been pricked in the intergyral spaces will sufficient fluid escape to allow the membrane to settle down closely over the pia—a matter of importance in cortical faradization to be emphasized later.

Once it has emerged from the ventricle and reached the surface of the brain, the fluid may leave the cranial and spinal chambers by forcing its way into the lymph spaces along the course of the nerves, and in this way may pass into the nasal membrane along the olfactory nerves, into the orbital tissues by the ophthalmic and optic, etc. as can be shown experimentally; but this method of escape is by far the least important. Most of the fluid passes directly into the sinuses—into the longitudinal sinus in particular. Key and Retzius long ago expressed the view that the escape takes place through the intervention of the Pacchionian granulations which project into the lacunæ laterales. Foramina of exit certainly exist in this situation, but whether the escape of fluid takes place through the active agency of these granulations may be doubted; for the free escape of fluid by the sinuses may be easily demonstrated in infants and in the higher apes, in whom no Pacchionian villi exist. Whether at the points of entry of fluid there exists some valvular structure akin to the valve where the thoracic duct enters the jugular or whether there be some other mechanism, matters not for our purpose; the essential thing is the fact that the chief escape takes place into the venous sinuses.

With this knowledge we may understand how infectious processes may block these channels and cause stasis of the cerebrospinal fluid; why thrombosis of the longitudinal sinus may lead to serious symptoms, even though the venous anastomosis may be sufficient to carry away the circulating blood; why congenital hydrocephalus is so frequently unassociated with any demonstrable lesion of the region of the foramen of Magendie; and finally why many of our operations directed toward the cure of hydrocephalus are based on wrong principles of drainage.

BRAIN.

Relation to skull.—The fully developed brain completely fills the cranial chamber, the configuration of which is determined under ordinary circumstances more by intracranial influences of growth than by adventitious extracranial influences. The shape of the brain, it is true, may be modified by long-continued pressure against the skull, as practiced in certain barbaric tribes, but it is doubtful whether the total quantity of brain can be modified by any such influence. Experimental attempts on animals to restrict cerebral growth by long-continued extracranial pressure have been unavailing. This matter has an important bearing upon a form of treatment (linear craniectomy) proposed for cases of cerebral maldevelopment, on the view that microcephalus is due to a primary closure of sutures which prevents cerebral expansion, rather than to a primary insufficiency of brain mass. It may be noted in this connection that local cerebral defects—whether from interrupted growth or from destructive lesions which have occurred at birth or during infancy—often-times indicate their presence by the flattening of the overlying portion of skull. Contrariwise, the cranium in the young, even long after the sutures have closed, yields quickly to abnormal growth or increase in size of the brain, as seen in cases of tumor or hydrocephalus.

Furthermore, it must be remembered that normal variations are great not only between the brains of individuals, but between those of different races. In individuals, for example, there may be a relatively great disproportion between the amount of brain matter anterior and that posterior to the central fissure, and Frohriepp refers to two types—the “frontopetal,” in which the greater portion of the brain lies anterior to a perpendicular line erected at the auditory meatus, and the “occipitopetal,” in which it is posterior to this line. And in these two forms not only the position of the Rolandic fissure, but its angle of obliquity as well, is greatly altered. There is probably a still greater variation between races. Bean has pointed out the great difference in the size and shape of the Caucasian and Ethiopian brains, particularly as regards the frontal lobe. It is in consequence of these things that no accurate extracranial measurements may serve to indicate, other than roughly, the situation and the form of the central or other cerebral fissures.

The blood-supply.—The chief arterial stems, which have a free intercommunication through the circle of Willis, supply the hemispheres through three main branches: (1) The anterior cerebral supplies the first and second frontal convolutions and all of the mesial surface back to the parieto-occipital fissure; (2) the middle cerebral,

emerging at the Sylvian fissure, supplies the insula, the third frontal, the pre- and post-Rolandic convolutions, the parietal lobe, the first and second temporal convolutions, and part of the occipital lobe—in other words, most of the exposed outer surface of the hemisphere, and (3) the posterior cerebral supplies the second and third temporal gyri, the mesial part of the occipital lobe, and the under surface of the temporosphenoidal lobe. The vessels to the deeper structures are given off directly from the circle of Willis, or else from these main stems; those of chief moment penetrate and supply the basal ganglia. There are also three branches of considerable size which arise from the vertebral and supply the cerebellum.

The occlusion of any one of these large vessels gives characteristic and localizing signs, and operative ligation of the larger stems near the base of the hemisphere must be undertaken with caution, lest it lead to softening of an extensive cerebral area. It is for this reason that in operations for tumors it is desirable to ligate the individual twigs in the environ of the growth, rather than the main branch at a distance. This applies also to ligation of the carotid, for in spite of the usual free anastomosis at the circle of Willis, extensive softening may follow this operation.

The *venous* circulation within the brain itself and over the cortex is also important. Of chief interest is the peculiar arrangement of vessels which, collecting blood from the basal ganglia and from the choroid plexus, unite in the *venæ Galeni*, which in turn empty into the sinus rectus. Compression of these latter vessels, or their occlusion from any cause, leads to serious symptoms of stasis and to internal hydrocephalus.

The vessels of the hemisphere course over the surface and empty, for the most part, into the superior sagittal sinus by long, oblique passages directed forward; that is, against the current in the sinus. The two largest and most important of these superior cerebral veins lie in or near the central or the two adjoining sulci; they communicate freely with the large veins lying in the Sylvian fissure. Similar large vessels radiate from the temporo-sphenoidal lobe and enter the lateral sinuses. The points of attachment or anchorage of the hemispheres to the dura at the points where these large collecting veins cross the subdural space are of prime surgical importance; they occur chiefly at the parasinoidal sinuses a centimeter or two from the mid-sagittal line; also at the occipital pole, under the temporal lobe; and there is in addition a point of anchorage at the outer side of the cerebellar hemisphere.

It is undetermined whether the cerebral vessels possess *vasomotor nerves*. By intravital methods of staining it is possible to demonstrate nerves which are histologically akin to the vasomotor nerves

of other parts of the body, but they have never been unequivocally shown to possess a dilator or constrictor function. Certainly from the results of experimental observations it may be said that, even though vasomotor nerves be actually present, their physiological action differs from those to the splanchnic field under the control of the vasomotor center in the medulla.

The *lymph circulation* in the brain, though doubtless abundant, is of a peculiar nature not well understood. It has been demonstrated that the cerebrospinal fluid—which probably has no direct connection with the lymphatic system—escapes from the subarachnoid space directly into the larger sinuses and thus reaches the blood stream without the intervention of glands. Asher has shown that in all parts of the body lymph must become altered in its passage through glands before it enters the general circulation; for unaltered it is very toxic. It would be surprising should there prove to be an exception to this in the intracranial lymph circulation. It is possible that lymphatics from the surface of the brain may pass by channels through the cranial foramina into the external coverings and thus in the cervical glands without any actual communication with the subarachnoid space.

Physiology of cerebral circulation.—Certain general tenets of surgical import, for which we are in large part indebted to Leonard Hill's studies, may be mentioned:

The brain pulsates synchronously with pulse and respiration. Its greatest expansion is in expiration, due to the accompanying slight venous stasis. The cardiac pulse is transmitted to the cerebral veins. These movements are made possible by the ebb and flow of cerebrospinal fluid. Any increased tension of the dura mater decreases the exhibition of the cerebral movements.

As there is no evidence of the existence of a local vasomotor mechanism it follows that cerebral anemia from spasm of the cerebral arterioles does not occur, though it must be confessed that clinical evidence furnished by cases of Raynaud's disease, in which local cerebral symptoms occur, speaks against this view of the experimentalists.

According to the "Monro-Kellie doctrine" the total quantity of blood within the cranium is, under all physiological conditions, practically invariable. The amount of blood which passes through the cerebral vessels in a given time does, however, vary in wide limits.

Under normal circumstances the intracranial pressure may vary considerably [between 0 and 50 mm. of Hg. (Hill)] from circulatory alterations alone, brought about by changes in position, straining, etc. Venous congestion, when kept up, may, however, become of great pathological significance.

Conditions of disease, on the other hand, leading to the presence of a new body in the brain—a clot, a tumor, hydrocephalus, etc.—materially affect the amount of blood in the brain. They lead primarily to venous congestion, and only to arterial anemia when the pressure due to the crowding of the new body exceeds the general arterial pressure. Even under these circumstances anemia is for a time overcome by a compensatory rise in arterial tension, due largely to constriction of the splanchnic field. The cerebral sinuses are, in a measure, compressible.

Under many circumstances gravity plays an important rôle in cerebral circulation, the vasomotor splanchnic mechanism being the regulatory agent and one which is more perfect in its action in upright animals than in those normally on "all fours." Inefficiency of the splanchnic constrictors due to injuries, to chloroform, etc., is of vital importance, as the lowered pressure lessens the cerebral circulation and leads to anemia. Hence the feet-down position when the splanchnic compensation fails may lead to a cessation of cerebral circulation, with fainting and even death. Some form of support, like Crile's rubber suit, alone will justify this position when the vasomotor system is affected.

In cerebral anemia, whether due to vasomotor paralysis and posture, to blood-letting, to occlusion of important vessels by operation or disease, symptoms occur which are comparable with those of asphyxia. There is first unconsciousness, followed by slow pulse and rise in blood pressure; later by a fall in blood pressure, a rapid pulse, and death.

The rapid occlusion of the main source of arterial blood (the two carotids) is likely to lead to fatal symptoms of anemia; their gradual occlusion can safely be carried out by some such measure as Halsted has described.

The tension of the cerebrospinal fluid and cerebral venous pressure is normally the same. If the former is increased over the latter the fluid escapes into the sinuses (unless they are occluded). If the venous pressure becomes greater than the cerebrospinal fluid tension there is a damming back of cerebrospinal fluid.

Localization of function.—It was the teaching of Flourens that all parts of the cortex possessed the same significance; his view being that lesions of the hemispheres would produce depression of function merely according to their extent, but regardless of their situation. Thus, the phrenologists, Gall and Spurzheim, with their somewhat visionary hypotheses, were, in principle, nearer the truth in their territorial subdivisions than their more distinguished contemporary. From clinical observations, Broca, in 1861, by demonstrating the seat of articular language; Hughlings Jackson, in 1864, as a result of the study of focal epilepsy and Bastian, in 1869, through further observations on disturbance of speech, undermined in a measure the doctrines of Flourens; but they did not completely crumble to the ground until 1870 when my experimental methods Fritsch and Hitzig demonstrated that there were areas of the dog's cortex which gave excitomotor responses to galvanic stimulation. Ferrier, in 1873, using the faradic current, verified and amplified their observations, and the subsequent studies of Burdon Sanderson, Munk, Schiff, Schäfer, Horsley, Mott, Bianchi, and a host of others have served to fully establish the theory of separate localization of cortical function. The results of these clinical and experimental researches have lately received further confirmation by embryological and histological studies, notable among which are Flechsig's observations on the periods of myelination of the separate tracts, and the investigations by Mynert, Betz, Ramon y Cajal, and more recently by Campbell, which have thrown light on the structural differences of the various cortical areas.

The localization theory, however, was not received without opposition on the part of many clinicians and experimentalists, not a few

of whom, like Goltz, were led to interpret their experimental observations as an argument against the localization of function. Even among those who supported the theory in general, disagreements have occurred in regard not only to the delineation, but also to the exact function of particular areas. The chief strife has been waged over the so-called sensorimotor cortex in the effort to determine whether there was a separate or a superimposed field of representation for sensory perception and motor discharge. Doubtless the chief reason for adherence to the view of superimposition of these areas was due to the fact that lesions of the supposed motor field, at a time when motor centers were thought to lie both anterior and posterior to the central fissure, often led to sensory disturbances. We now know, through the more accurate methods of cortical stimulation introduced by Sherrington and Grünbaum (1901), that that portion of the cortex which is directly excitable by a unipolar electrode consists of a narrow strip which lies anterior to the central fissure and extends to the depth of this fissure on its anterior surface alone. This circumscription of true "motor cortex," together with the subsequent histological demonstration that this zone corresponds exactly to the distribution of the Betz cells, has finally led to the general view that the central fissure divides the cortex into an anterior motor and a posterior sensory field, intricate though the commissural connections between these two fields may be. Sherrington and Grünbaum's observations on the higher anthropoids have been confirmed for man by Krause, Frazier, and the author.

The excitomotor cortex.—This is limited to a narrow strip, 1 centimeter or more in width, of the exposed part of the *gyrus centralis anterior*, but extends to the depth of the *fissura centralis* (*Rolandii*). Hence its chief portion is not on the visible surface, and consequently a lesion which actually involves the motor cortex may lie far below the exposed surface of the hemisphere. The anterior edge of the excitable area shades off without sharp demarcation; its upper limit overlaps slightly onto the mesial surface (*lobulus paracentralis*) and its lower limit falls short of the Sylvian fissure.

The Rolandic fissure is not a straight line, but is broken by two, or sometimes three, more or less well-developed angles (*genua*), formed, I believe, by the swellings above and below them, made by the aggregations of cells controlling movements in leg, arm, face, and, still lower down, jaws, tongue, etc. Opposite to the upper two *genua* the motor strip is less wide and its representative movements less complex, occurring as they do in neck and trunk. Thus, the *genua* are valuable surgical landmarks, particularly the middle and inferior ones, for they are more often brought into view. Above the superior *genu* there is but a small triangle of motor cortex which can be exposed, and it gives, on stimulation, movements in hip, knee, and toes;

opposite to this genu lie centers for movements of thorax and abdomen; between it and the middle genu lie centers for the upper extremity, the shoulder being represented higher than fingers and thumb; opposite to the middle genu are centers for the neck and below it those of the face—eyelids above and lips below; centers for jaws, tongue, vocal cord, pharynx, etc., are still lower, usually below an inferior genu.

Extirpation of these areas leads to loss of movement, which is more or less complete and permanent according to the totality of the extirpation and to the degree of bilateral representation of the particular movements concerned. Sensation is not affected.

Certain complex movements of a higher order may be obtained by simulation of areas adjoining the true motor cortex. Thus, below the *gyrus centralis anterior*, in the *pars opercularis*, sucking, chewing, sneezing, and vocalizing movements may be obtained (note that this is near the vocal speech center of Broca); and from the *gyrus frontalis medius* movements of the head and eyes to the opposite side may be elicited.

The pathway from the motor cortex is the pyramidal tract, whose fibers degenerate throughout their full length after injury to their cortical cells.

The sensory field.—It has long been known that lesions near the *fissura centralis* often lead to sensory disturbances. The observations of Sherrington and Grünbaum, showing that the posterior confines of the motor cortex lie in the floor of the fissure, paved the way for further study of the *gyrus centralis posterior*, and Campbell's researches in particular would seem to show that the primary registration of "common sensation" occurs there. He has demonstrated that histological changes are found in the cortical cells of this gyrus after amputations, in tabes, etc. The area occupies much the same position posteriorly in regard to the *fissura centralis* that the motor area holds anteriorly. It is largely hidden from view on the posterior surface of the fissure and does not extend back over more than the anterior half of the exposed postcentral gyrus.

I have had occasion to learn, from extirpations of the postcentral gyrus in cases of focal epilepsy with a sensory aura, that temporary sensory disturbances of the cortical type occur after such lesions. It is to be noted, furthermore, that some disturbance of motion follows such extirpations, but a palsy of this type is due to loss of afferent impulses and is unassociated, as I have found in two cases, with any degeneration of the pyramidal tract.

The fibers to the sensory field pass from the thalamus in the "cortical lemniscus" (Monakow) of the *corona radiata* to the post-Rolandic territory. In their course they lie in the posterior part of the *capsula interna*.

The forms of sensation, registration of which we may now, with some assurance, place in the near postcentral region, are the tactile sense, the muscular sense, and the power of discriminating points in contact. It is evident also that as one goes further back from the *fissura centralis* and approaches the posterior association field of Flechsig, sensation becomes more complex, so that more extensive and deeper lesions are necessary to interrupt its transmission. The senses of pain and of temperature lie probably in the intermediate post-central zone of Campbell and that for the recognition of objects—the stereognostic sense in particular—is located as far back as in the parietal lobe.

The visual cortex.—Practically all investigators agree in placing the primary receiving station for visual impressions in the occipital lobe, particularly on its mesial surface in the calcarine region. The investing (visuo-psychic) field is concerned with “the final elaboration and interpretation of these sensations.” Myelinization of the fibers to the former occurs early; of those to the latter, not until the child is capable of interpreting visual stimuli.

The visuo-psychic field extends on the outer surface (of the left side) in the second occipital convolution as far as the angular gyrus, where lies the visual word center (reading) which participates in the speech mechanism. The lingual lobule below the *fissura calcarina* appears to be associated with color perception.

The auditory cortex.—Auditory impulses appear to be received primarily at some portion of the *gyrus temporalis superior* and to be “converted into conscious perceptions” in adjoining parts of the temporal lobe, those on the left side in particular being concerned with the auditory end of the speech mechanism. Extensive lesions on the right side may give rise to no appreciable impairment of hearing on the same side, and there is much confusion over the unilaterality or otherwise of the registration of auditory impulses.

The elaboration of the primary stimuli into tone perception, word perception, etc., occurs in the outlying districts, namely, in the audito-psychic area which envelopes the primary receiving station.

The olfactory cortex.—The *lobus pyriformis* is generally regarded as the chief cortical center for olfaction, but there is a division of opinion as to the part played by the adjoining areas of the *gyrus uncinatus*, *cornu Ammonis*, etc.

The *gustatory area*, like the above, is not definitely determined, but it also lies probably at the lip of the limbic lobe, in the neighborhood of the uncus. This, topographically speaking, would place both of these areas, for taste and smell, in a situation just to the outer side of the pituitary fossa—a matter of considerable importance, as lesions confined to this area of the limbic lobe not only give characteristic symptoms, but are surgically approachable.

The four cortical areas concerned in speech in right-handed people.—

(1) The center for the recognition of spoken words lies in the outskirts of the primary center for hearing in the *gyrus temporalis superior* of the left temporal lobe. This doubtless is the first center concerned in the development of the faculty of language, in normal individuals, at least, for we must remember that speech may be acquired primarily through the sense of touch as exemplified in Helen Keller's remarkable case.

(2) Since Broca, and until the recent doubts cast upon it by Marie, the posterior end of the *gyrus frontalis inferior* has, by common consent, been regarded as harboring the centers for motor or vocal speech. These auditory and vocal word centers—Wylie's "primary couple"—may be developed in the uneducated with but little further advance. With later education is acquired the interpretation (reading) and the making (writing) of the symbols of language.

(3) The visual word center concerned in reading has been definitely placed in the *gyrus angularis* in the outskirts of the visuo-psychic field, and—

(4) The so-called writing center, if such exists, has been placed at the posterior end of the *gyrus frontalis medius*; in other words, near the primary centers for movements of the hand and fingers. It is not improbable also that there is a fifth center in the parietal lobe, associating the sense of touch with the speech mechanism. It is to be remembered that no part of the speech mechanism, so far as its cortical centers are concerned, can be upset without affecting in some degree all other parts, though the most serious disturbances result from lesions of one of the "primary couple."

The association fields.—Other parts of the cortex than those which have been described are concerned, so far as is known, only with the complex processes of association, and lesions of these areas are largely "silent," so far as our present possibilities of neurological examination go. An exception may be made in the case of the frontal lobes, particularly the "prefrontal" portion of the left hemisphere, where the higher psychic or intellectual faculty has been placed by many observers.

THE SYMPTOMATOLOGY OF ORGANIC LESIONS.

Although the cerebrum is subject to a great variety of morbid conditions, the symptoms which are manifested thereby are relatively few. In diseases of the nervous system they depend in general more upon the situation of the process than upon its nature. Thus, an obstructive lesion of the motor path, at any point between the cortex and the peripheral end-organs, results in paralysis, whether it be due

to a neoplasm, a vascular lesion (hemorrhage, thrombosis, or embolism), inflammation, injury, compression, or what not.

In a broad sense the symptoms may be divided into (1) *subjective* symptoms, appreciated only by the patient, and *objective* ones, which are obvious to the observer; (2) *general* symptoms, or those which characterize many intracranial processes regardless of their seat, and *focal* ones, which indicate the situation of the lesion. Subjective or objective symptoms may be general or focal, and vice versa; general or focal symptoms may be either largely subjective or objective. For example, a tumor involving the cortical center of the left hemisphere, which presides over movements of the arm, may give subjective symptoms which are general and due to pressure, such as diffuse headache, nausea, or dizziness; or it may give others which are focal, as weakness or diminished sensibility in the arm. It may give objective symptoms which are general, as shown by vomiting, choked disk, and slowed pulse, or ones which are focal, such as muscular atrophy, brachial spasticity, or a monoplegic spasm.

The symptoms, broadly speaking, may further be divided into those which are *irritative* and those which are *paralytic*. These again may be general or focal, subjective or objective.

General symptoms.—These are headaches, vomiting, choked disk, and other evidences of venous stasis, vertigo, convulsions, etc.

Headache.—This is of common occurrence (1) in association with a variety of conditions not primarily cerebral; but a patient subject to persisting headaches should rest under the suspicion of having primary intracranial disease until this can be definitely disproved. Cephalalgia of extracranial origin accompanies chronic processes in the mastoid or accessory sinuses, eye strain, etc. (the so-called reflex headaches), anemia, disturbances of digestive or menstrual function, fevers, etc.

Headache (2) is almost invariable in diseases of the meninges, particularly when the sentient dura is involved, and it is my impression that most headaches—those of the migrainous as well as of other types—are largely dural in origin. This membrane is innervated entirely by the trigeminus, with the exception of a small area about the foramen magnum supplied by vagal fibers. As the recurring pain of “hemicrania” is a not infrequent antecedent of facial neuralgia, this may possibly be considered as a form of dural (trigeminal) neuralgia. The headaches of meningeal inflammation, particularly of the luetic type, may be profound.

Of chief importance, however, are the headaches (3) due to intracranial pressure from any cause whatsoever. In tumor, edema (whether of traumatic origin or associated with nephritis), internal hydrocephalus, serous meningitis, etc., the pain is doubtless brought about by abnormal tension of the dura or of its membranous ex-

pansions into falx and tentorium. As a rule, headaches due to pressure are of no particular help in localization, for regardless of the seat of the lesion, they may be referred to the vertex, to the frontal region, or to the occiput. Occasionally, however, taken in conjunction with other symptoms, their situation may be helpful—as in certain subtentorial lesions in which the chief discomfort may be referred to the corresponding suboccipital area. This is especially true of local headaches which are associated with an area of tenderness.

There are all gradations of headache, from a dull sense of pressure or fullness to agonizing and prostrating pain.

Vomiting, with or without *nausea*, and irrespective of any gastric disturbance, is a common symptom of any acute or chronic cerebral lesion, especially of those which encroach upon the intracranial space. It may be an early symptom of concussion or contusion; and more or less nausea, doubtless due to the secondary edema, may persist after these acute traumatic lesions for days. In chronic processes it is an even more characteristic symptom. Sudden projectile vomiting, often accompanied by nausea, is well known as a general symptom of pressure resulting from brain tumor, from the edema of nephritis, etc. The physiology of the process is not determined. Some believe that there is an especial center in the medulla, irritation of which leads to vomiting.

Vertigo is a frequent subjective phenomenon, but being a common symptom from causes other than cerebral ones it is of importance only in association with other evidences of organic disease of the brain. It is most pronounced in lesions which involve the auditory nerve, the mid-brain, or cerebellum.

Choked disk ("optic neuritis"), if of a degree sufficient to cause dimness of vision, may be a subjective as well as an objective phenomenon. It is one of the most important indications of intracranial pressure, and an ophthalmoscopic examination should be made upon every case in which, from headache alone, there may be a suspicion of intracranial disease. It is not sufficient for the examiner to be able to recognize a choked disk when it is full-blown, but the slight edema of retina and nerve head with early distension and tortuosity of the veins which precedes actual "choking," must be appreciated, for they are of the utmost help in making an early diagnosis. The symptom, on the whole, is merely an evidence of general pressure, although occasionally a unilateral choking or a process more advanced in one side than the other may have a certain localizing value and suggest the presence of disease upon the corresponding side of the head. As a matter of fact, however, the most pronounced cases of choked disk occur in association with subtentorial processes, the stasis in the eye grounds being brought about by an obstructive

hydrocephalus; and, inasmuch as this leads to a general and equal increase of pressure the process on the two sides will be equal in degree, unless there chances to be some structural difference in the sheaths of the two optic nerves.

Many theories have been advanced in explanation of this phenomenon. The more important views are those of von Graefe (1860), who thought that it was due to compression of the cavernous sinus leading to venous stasis in the eye. The Schmidt-Manz "*Transporttheorie*" (following the discovery of Schwalbe that the intravaginal space of the optic nerve communicates with the intermeningeal spaces of the brain) attributes a choked disk, in cases of increased pressure, to the stasis of cerebrospinal fluid into this space, leading to an ampullalike distension of the optic sheath. Parinaud (1879) thought that a choked disk could only occur in association with internal hydrocephalus or other conditions associated with an edema, which was supposed to spread along the optic nerve from the brain itself. Von Leber (1881) expressed the view that it was an actual inflammation—a "papillitis"—brought about, irrespective of edema or stasis, by the growth; just as a "neuroretinitis" is said to be caused by circulating toxic products in chronic renal disease. Other theories—that it is of reflex origin; that it is due to sympathetic disturbances, etc.—have been advanced, but they are less widely supported.

Generally speaking, we see that there are mechanical views opposed to toxic views, each of which has been upheld by a number of eminent clinicians and investigators; but the general employment of the term optic "neuritis" would seem to indicate that it is widely regarded as an inflammatory process. Recent clinical and experimental observations showing the rapid subsidence of a choked disk after decompressive operations, serve to modify the views which many have held, and with Sanger, Axenfeld, and others I believe that almost all, if not all cases of choked disk are primarily of mechanical origin and do not justify the term "neuritis." It is a quibble to say that the transudation of fluid anywhere is necessarily associated with some toxic agency, but the stasis edema which occurs in consequence of the application of a tourniquet on an extremity, though toxic in a sense, is more properly considered mechanical, since the removal of the mechanical agent and relief of the stasis allows the edema rapidly to subside.

When the swelling of a choked disk has become pronounced and has been of sufficiently long duration hemorrhages occur in the nerve head and retina as a result of the stasis; this is followed by an infiltration of round cells, and unless the process is checked organization progresses until the fibers become physiologically "blocked" and light is no longer transmitted. The loss of vision occurs primarily

as a peripheral shrinkage of the field for form and color, and it must always be remembered that normal acuity of central vision may remain oftentimes until late in the process.

Other less important general symptoms may be mentioned, among them *convulsions*, which in this case are invariably associated with loss of consciousness. They may occur even when there is a local lesion and yet not be indicative of its situation, for only when the lesion involves primarily some center, irritation of which leads to symptoms which can be appreciated by the patient during conscious moments or can be observed by the onlooker before the convulsion becomes general, can their seat of origin be determined. Focal lesions, therefore, apart from the so-called motor fields, may lead to a general rather than a focal convulsion. General convulsions of cerebral origin involving the entire musculature may arise from toxic causes apart from any organic lesion; such are especially common in children.

Grades of stupor are usual in almost all intracranial processes, especially when acute or subacute. They may vary from simple drowsiness, with yawning, etc., to profound sleep from which the patient can not be aroused; to lethargy, in which he is totally indifferent to his condition, even though seemingly awake; to stupor, in which he is oblivious of his surroundings, though in its lighter grades he may be aroused so as to respond to questions, of which he has no subsequent memory; to coma, with profound unconsciousness and usually with serious respiratory symptoms. Corresponding degrees of unconsciousness may be of toxic origin and occur in acute alcohol poisoning, in diabetes, etc., or they may be due to circulatory disturbances, whether from acute anemia or from the venous stasis brought about by intracranial pressure. They not only accompany all cranial injuries of any severity, due to the pressure of hemorrhage, edema, etc., but they also occur in meningitis and internal hydrocephalus as well. When unconsciousness is profound and results in coma it is clinically of great importance to distinguish between its possible sources of origin—apoplexy (whether spontaneous or traumatic), sunstroke, alcoholism, uremia, narcotic poisoning, diabetes, epilepsy, etc.

Insomnia, emaciation, variations from the normal of pulse, of respiration, of body temperature, of urinary secretion, may be regarded as general symptoms in certain cases.

Focal symptoms.—We have heretofore considered merely the general symptoms which indicate the presence of intracranial disease. When focal symptoms are present they may enable us to determine its situation. Roughly speaking, they are confined to disturbances of motion, of common sensation, and of the faculties of special sense.

On the motor side.—Motor paralysis is the most evident of all objective signs. Primarily it indicates the side of the brain involved. Less clearly it shows the situation of the involvement, whether cortical, subcortical, capsular, penduncular, pontine, or medullary. It may be hemiplegic and involve an entire half of the body, or only the trunk and extremities. It may be monoplegic and involve only one extremity; paraplegic when the legs chiefly are affected; or diplegic when arms and legs both are impaired. Diplegia is a more usual evidence of spinal than of cerebral disease, though it occurs in cases of widespread intracranial hemorrhage or injury, particularly in the so-called “birth palsies.” There are also various forms of multiple paralysis, in which individual muscles or groups of muscles supplied by single cerebral nerves may be involved.

Paralyses of cortical origin are apt to be accompanied or preceded by irritative symptoms, and are more commonly monoplegic in character. Paralyses of capsular or peduncular origin, since the fibers of the pyramidal tract are gathered there into a small space, are more apt to be hemiplegic. The so-called “crossed paralysis” is one in which a cerebral nerve palsy on one side accompanies paralysis of the limbs on the opposite side. Motor paralyses of pontine or medullary origin rarely fail to have accompanying symptoms, due to involvement of neighboring structures.

Spasticity of the muscles, with increased reflexes, occurs as the result of a lesion of the intracranial portion of the motor pathway. Should the lesion take place in the young there may be marked disturbance of growth. Contractures occur, whereby the limbs become fixed in awkward positions.

Motor irritation is evidenced most frequently by epileptiform seizures or convulsions. Local convulsions in the form of monospasm is a common indication of a lesion at or near the so-called motor area. The process leading to convulsions may be a quiescent one—a cortical defect; the cicatrix of an old healed focus of hemorrhage, etc.—or one which is progressive, as an enlarging cyst or tumor. A localized convulsion may be followed by paralysis; a general one, by a condition of profound muscular exhaustion; and when frequently repeated and the so-called status epilepticus ensues, death may result from asphyxia due to failure of respiratory movements. Other irregularities of movement—ataxic, choreic, athetoid, etc.—are usually the result of lesions in organs like the cerebellum or basal ganglia which modify movements, rather than of those affecting the primary conducting path.

On the sensory side.—Like those of motion, sensory disturbances may result from cortical or subcortical lesions. They also may be irritative and associated with subjective symptoms of paresthesia, or paralytic and accompanied by anesthesia.

Anesthesia may be complete or partial (hypesthesia) to various forms of stimulation—to pain, touch, pressure, temperature, etc. The deeper, as well as the cutaneous sense, may be affected and there may be loss of the sense of posture in an extremity, or of its position in space. Owing to the fact that a profound sensory paralysis leads to the shutting off of all afferent impulses, a certain degree of motor impairment is an almost invariable accompaniment. On the other hand, no sensory disturbance need accompany motor paralysis.

Irritative sensory symptoms may precede those of motion. Thus, the aura or warning of an impending convulsion may be an important clinical sign and may at times serve to indicate the situation of the lesion, even though the convulsion itself was general from the onset. The warning may occur as a subjective sensory, gustatory, visual, or olfactory impression.

Disturbances of *special sense perception* may be secondary not only to peripheral lesions of the nerves themselves, but also to lesions of the cerebral centers where special sense impressions are registered. A lesion may pervert or destroy any of the special sense qualities—smell, taste, hearing, and sight.

Craniocerebral topography.—The discovery that different functions were localized in particular areas of the brain, and the knowledge—gradually acquired—of what symptoms were produced by lesions limited to these areas, paved the way for surgical measures directed toward their relief. It thus became imperative, particularly in the days when an attempt was made to approach these lesions through a small trephine opening, that the relationship of the cerebral convolutions and fissures to the surface markings of the skull should be established with the greatest possible accuracy. The science of craniocerebral topography arose; and a great number of investigations have since been devoted to the establishment of certain rules of measurement, which give us, with sufficient accuracy, the average position of the main fissures of the exposed part of the hemispheres in their relation to definitely palpable points on the external cranial wall.

It may be recalled that Paul Broca was not only a pioneer in these studies, but was actually the first, in 1871, to put them to practical test in an operation performed for him on a patient with aphasia due to an abscess in the third left frontal convolution. Since then important contributions to the subject have been made, and particular rules have been formulated by Reid, Cunningham, Thane, Horsley, Chipault, Taylor and Haughton, Poirier, Dana, Krönlein, Chiene, Anderson and Makin, Le Fort and Debierre, Masse and Woolingham, Lannelongue and Mauclair (for infants), Müller, Froriep, Kocher, and many others. Each of these prescribed rules has its good points, and the results of most of them, when applied to the same skull, do

not vary much more than a centimeter or two. For the most part they are devoted to a determination upon the scalp of the upper and lower ends of the central fissure (superior and inferior Rolandic points), to the point of origin of the Sylvian fissure (Sylvian point), as well as to its line of general direction, and, less important, to the occipitoparietal fissure. The studies have been based largely on the average measurements of adult crania, and the difficulty of establishing the exact relationship of the encephalic and the extracranial landmarks has been overcome by a variety of ingenious devices.

The main principles of the superficial delineation of the fissures rest (1) upon the establishment of an equatorial base line, from which perpendicular meridians or lines of intersection of coronal planes are erected at given points, and (2) upon angulation at given points, either from this base line or from parallel circles, or from the midsagittal meridian. Reid's base line is the favorite among English writers; it passes through the lower border of the orbit and middle of the external meatus and is nearly parallel to the upper border of the zygoma; the German base line differs slightly from this in passing through the upper edge of the meatus. Perpendiculars according to various rules of measurement are erected (1) at the preauricular point between tragus and maxillary condyle; (2) from this condyle itself; (3) from the middle of the zygoma; and (4) from the posterior edge of the mastoid process. Zones parallel to the base line ("upper horizontals") are made to pass through the upper border of the orbit, or from the external angular process, etc. Use is made also of particular points of union of the sutures, many of which are more or less definitely palpable and for which time-honored names are preserved—nasion, inion, pterion, bregma, asterion, etc.

A few of the better rules for the extracranial determination of the chief cerebral fissures may be given.

The superior Rolandic point is found on the midsagittal line 55.6 per cent of the distance from glabella to inion or external occipital protuberance (Reid); or one-half this distance plus one-half to three-fourths inches (Thane); or one-half the nasio-inionic line plus 2 cm. (Poirier), or $2\frac{1}{2}$ cm. (Kocher); or at the intersection of the midsagittal line with a coronal plane erected at the posterior border of the mastoid (Reid, Krönlein); or 5 cm. posterior to the intersection with a coronal plane erected at the preauricular point (Broca); or 2 to 3 cm. behind the coronal suture or bregma (Thane).

The inferior Rolandic point lies on the fissure of Sylvius 25 mm. behind its bifurcation or Sylvian point (Thane); or $3\frac{1}{4}$ inches below the superior Rolandic point on a line which makes an angle of from 67 to 71.5 degrees with the midlongitudinal line (Cunningham); or 7 cm. above the pre-auricular point on a line perpendicular to the

zygoma (Poirier); or 5.5 cm. (varying from 4 to 7) above the zygoma on or slightly in front of this pre-auricular line (Thane).

The *Rolandic line* corresponds with a meridian dropped from the superior Rolandic point and making an angle with the midsagittal line of 67 degrees (Hare); or varying from 64° to 75° (Thane). If extended below the inferior Rolandic point it should cross the middle of the zygomatic arch (Le Fort).

The *Sylvian point* may be located at the intersection of two lines; (1) from the auditory meatus to a point at 25 per cent of the nasio-inionic line, and (2) from the external angular process to a point at 75 per cent of nasio-inionic line (Taylor and Haughton); or at the intersection of lines (1) erected perpendicular to the base line at the middle of the zygoma and (2) parallel to the base line (upper horizontal) through the upper margin of the orbit (Krönlein); or 12 mm. above a horizontal line drawn back from the frontomalar suture to a distance of 35 mm. (Thane).

The *fissure of Sylvius* corresponds with a line joining the external angular process of the frontal bone and the point of intersection of the previously determined Rolandic line and the preauricular perpendicular line (Reid); or with a line drawn from the nasion to 1 cm. below the lambda (Poirier); or with a line connecting the external angular process with a point 80 per cent of the distance from nasion to inion (Chipault); or a point 75 per cent of this distance (Taylor and Haughton); or with a line bisecting the acute angle made by the lines (1) from the superior Rolandic point to the Sylvian point and (2) from the upper border of the orbit and carried parallel to the base line (Krönlein); or with a line from the external angular process to a point at the junction of the middle and lower thirds of the line connecting the preauricular point and midsagittal points (Anderson and Makins).

The *parieto-occipital fissure* is found opposite to or a little above the lambda, or 6½ cm. above the inion (Thane), or seven-eighths of the distance from the midsagittal point to the inion (Anderson and Makins).

Aside from these prescribed rules there are certain general points worthy of observation. The lower level of the temporal lobe—in other words, the floor of the middle cranial fossa—lies about on a level with the zygoma, and the lower edge of the occipital lobe corresponds with the superior curved line of the occipital bone. The Sylvian point, which marks the bifurcation of the Sylvian fissure, corresponds practically with the pterion, and the posterior arm of the fissure in the adult underlies the anterior part of the parietosquamosal suture and ascends to a point just below the parietal eminence; it consequently is much higher than one, not having made measurements, would suppose, for the center of the fissure is fully 2 inches

above the zygoma. The motor strip lies more on the top of the hemisphere than on the side and is entirely under the parietal bone; hence diagrams which, on a lateral view, show much more than the face centers—that is, the part below the middle genu—are incorrect and confusing. The mid-point between inion and nasion is easily determined and practically corresponds with a perpendicular erected from the base line at the meatus. Though this lies 1 or 2 cm. anterior to the superior Rolandic point, a meridian at 60 degrees dropped from this point to the middle of the zygoma, as Kocher has shown, indicates the general direction of the top of the precentral convolution; and this meridian—in view of Sherrington's finding that there is no motor cortex posterior to the central fissure—is as simple and useful a topographical landmark as can be had for general purposes.

It may further be noted that the Sylvian point marks the pole of the insula; that the parietal eminence overlies the supramarginal gyrus; the frontal eminence, the second convolution; the antero-inferior angle of the parietal, the inferior frontal (Broca's) convolution; that the temporal lobe lies for the most part beneath the squamous wing of the temporal bone; the parietal lobe entirely under the parietal bone.

As to *the deeper structures*, the Sylvian point being the guide to the insula, this in turn covers the basal ganglia, as has been pointed out when speaking of its early formation. The lateral ventricles curl round the basal ganglia with their flat surface on top at a depth of about 5 cm. below the upper surface of the hemisphere. If they are to be approached for aspiration it is desirable to select not only a site where little harm can be done from the passage of the needle, but also where there is the least likelihood of missing the ventricle. Paths of election, therefore, are (1) the superior frontal convolution to the area where the ventricle is horizontally wide over the basal ganglia, or (2) through the posterior end of the superior temporal convolution to the area where the cavity is vertically wide—namely, as it curls around the ganglia and gives off its temporal and occipital cornua.

The *middle meningeal artery*, after entering the skull at the foramen spinosum, curves forward and upward on the dura, covering the tip of the temporal lobe, to the antero-inferior angle of the parietal bone, which it deeply grooves or channels. The pterion, therefore, is a guide to the vessel in this part of its course where it overlies the Sylvian point; but for purposes of ligation the vessel can be exposed more safely by trephining in the middle of the temporal fossa, where it is easily approached. The attachment of the vessel in the bone at these two fixed points is to be observed in certain extradural operations in the middle fossa, as in the approach to the Gasserian ganglion.

It can be seen that the establishment of the position of the Rolandic fissure has been the chief aim of these investigations—an evidence of the fact that a large proportion of intracranial operations have been directed toward central lesions which loudly call attention to their presence by paralyses or convulsions. However, even in this carefully studied region—the most approachable one for the surgeon—extracranial measurements are not to be implicitly relied upon as guides for the recognition of the central convolutions, and the supplementary acquaintance with actual cortical topography is of far greater importance. Too great dependence on the former may be very misleading. Thus, though the superior Rolandic point can be determined upon the scalp with a small margin of error, this information is not of especial value, for the upper end of the central fissure is so inaccessible, owing to the parasinoidal sinuses, etc., that it can rarely be exposed. Furthermore, the fissure makes such a variable angle with the median line (64 to 75 degrees) and is so sinuous in its downward course, owing to the variable prominence of the genua, that even though the superior and inferior Rolandic points have been accurately determined, the line connecting them at the prescribed angle (averaging 67 degrees) may lie from 1 to 2 cm. (the width of a convolution) anterior or posterior to that part of the central fissure, which is usually brought into view by the operation.

These things are mentioned, not to deter surgeons from studying craniocerebral topography, but rather to point out that topographical delineations on the scalp are at best only a rough indication of cortical landmarks and that the ability to recognize these after exposure in the living is, after all, the essential thing. This presents little difficulty so far as the Sylvian fissure is concerned, and comes with practice in the case of the central fissure, though the final appeal must often be made to faradization. All neurological surgeons, through abundant practice on the cadaver, should acquire so thorough an acquaintance with the precepts of craniocerebral topography that they possess the ability to visualize through the skull not only the surface markings, but also the deeper structures of the encephalon, unaided by elaborate extracranial measurements. They should know the brain as abdominal surgeons know the belly.

INFLAMMATION OF THE BRAIN.

Acute encephalitis—*Polio-encephalitis acuta* (Strümpell).—A more or less diffuse inflammation of the cerebral cortex may occur (*a*) in certain toxic conditions due to gas poisoning, alcohol, etc.; (*b*) as a consequence of trauma; (*c*) as a complication of such acute infectious diseases as typhoid or influenza; or (*d*) with local suppurative processes, particularly those of the middle ear. The anatomical

features of the process are analogous to those which occur in the cord in acute poliomyelitis.

The general symptoms are those which accompany all severe acute cerebrospinal affections, and are due to tension—headache, somnolence, vomiting, fever, delirium, rapid pulse, and, in some cases of greater severity, choked disk, coma, with slow pulse, and respiratory changes. The local symptoms are variable and depend upon the situation and extent of the lesion. Paralyzes may occur or irritative symptoms of an epileptiform character.

Cerebral abscess.—An abscess in the brain is rarely, if ever, primary, although occasional instances have been recorded in which, even after a most detailed post-mortem examination, it has been impossible to attribute its presence to any external source (Mills and Spiller). It must be appreciated, however, that the lesion may be of such long standing that the original focus of suppuration elsewhere in the body may in the interval have completely healed.

It is usually a secondary process, and the three most important causal factors are: (1) *Trauma*, which accounts for 50 per cent of the cases. We have seen that it is a common sequel of penetrating wounds or fractures of the skull, when infected material of one sort or another has been inoculated directly into the brain substance. Although Bergmann expressed the opinion that it never follows a simple contusion of the head, there seem to be, nevertheless, authentic cases in which, in the absence of fracture or external evidence of suppuration, such a sequel has occurred (Ehrenrooth). In all probability the blow in these cases has led to a rupture of cortical vessels with extravasation, and there has been a subsequent hematogenous infection of the area of diminished tissue resistance thus produced, the clot proving a favorable soil for bacterial growth. It has already been shown that, in similar fashion, a local suppuration may occur in the cranial vault after a simple contusion; this in turn may lead to a cerebral abscess, often without any definite evidences of intervening meningeal infection. Symptoms of cerebral abscess may not appear until weeks or, indeed, months after a local wound or fracture of the skull has healed. This is especially true of cases in which a foreign body, such as a bullet or a broken knife blade, has been introduced at the time of the injury.

(2) In *pyemia*, the result of infected wounds, single or multiple abscesses of the brain can occur, but owing to the comparative rarity of this condition in later days they have become a relatively infrequent autopsy finding. Metastatic cerebral abscesses, however, may accompany ulcerative endocarditis, extensive osteomyelitis of the long bones, etc. Their association, particularly with suppurative pulmonic diseases, such as gangrene, empyema, or bronchiectasis,

has long been recognized. In 100 cases of pulmonary gangrene Nähter found 8 with cerebral abscess. Abscesses may follow the specific fevers, influenza, typhoid, etc., even without the association of otitis media.

Tuberculous abscesses—the solitary tubercle—are probably always metastatic. They will be considered under tumors.

(3) By far the most important group comprises those which occur from a more or less direct *extension of suppurative disease from the middle ear, mastoid cells, sphenoidal, or frontal sinuses*. As the result of the long-standing suppuration in one of these cavities a gradual necrosis of their thin protecting bony shell may take place. When necrosis has occurred any sudden flare-up in the activity of the disease may lead to involvement of the exposed meninges, and later of the brain itself, particularly if there be any tendency to retention of the secretions due to a cholesteatoma or exuberant granulations. Even with no intervening meningitis, the infection may be carried by thrombosed veins or along lymph-spaces directly into the subcortical tissue.

Focal meningeal abscesses also may occur without a spreading meningitis; they are more commonly extradural, though pial abscesses have been recorded by Randall, Spiller, and others.

The several *modes of infection*, therefore, are: (1) By direct inoculation; (2) by metastasis through the blood stream; (3) by direct extension of the suppurative process from extracranial cavities; (4) by inoculation through infected vessels which connect with an extracranial or extradural suppuration. In the latter instance, as already stated, there may be no visible connection whatever between the original focus and the subcortical abscess—a fact which often enhances the difficulty of the surgical problem. It has been suggested in explanation that the vascular leptomeninges and cortex offer a greater resistance to bacterial infection than the subcortical nerve tissue, to which the infective material is carried by the capillary lymphatic tubes which traverse the cortex at right angles to the surface and are in direct communication with the subarachnoid space.

Occurrence.—Abscesses of the frontal lobe most frequently follow infection of the frontal sinuses; of the temporal lobe infection in the middle ear or mastoid antrum; of the cerebellum the mastoid cells themselves, either direct or through an intervening sigmoid sinus phlebitis.

According to Grunert, 91 per cent follow chronic and only 9 per cent acute otitis media. Partly for this reason adults are more commonly affected. Holt, in his report of five cases in infants, calls attention to its presumed rarity in the young. Oppenheim has re-

ported instances of abscess formation after 34 and 45 years of chronic otitis media. I have seen cases after 26 and 30 years. In 9,000 autopsies at Guy's Hospital, Pitt found 56 brain abscesses, 18 of them of otitic origin, and only 1 due to nasal suppuration. In La Fort and Lehmann's statistics of 458 cases of abscess, the cerebrum was involved in 227; the cerebellum in 113; cerebrum and cerebellum in 11; pons, peduncles, or fourth ventricle in 7 cases. Thus we see that the cerebrum is affected more than twice as often as the cerebellum. Cassirer has reported 15 cases of the rarer abscesses which occur in the brain stem.

I have seen only two cases of frontal lobe abscess. One of them followed a chronic suppuration of the frontal sinus, attributed to a rhinological operation for the removal of a polyp—a not infrequent origin of sinus disease. The other was an acute case secondary to a bullet wound, the missile having traversed the base of the skull and orbit, opening and infecting the sinus. Neither of these cases recovered. In his study of the cerebral complications of sphenoidal sinus disease, St. Clair Thomson records but one case of abscess.

Morbid anatomy.—Abscesses may be solitary or multiple; diffuse or definitely circumscribed by a capsule; they are occasionally multilocular. The capsule may be rapidly formed (Starr), and the thickness of its wall may reach several millimeters. Abscesses of otitic origin may be bilateral (Whitehead). When multiple abscesses occur they are usually small; isolated ones sometimes reach such a size that they occupy the greater portion of a lobe.

The character of the pus varies greatly, depending upon the age of the abscess. In early cases it is mixed with the reddish débris of disorganized brain matter; in the later cases it is apt to have a greenish tint and a peculiarly disagreeable odor. The bacteriological examination may show a single or a mixed flora. Though staphylococci or streptococci are the ordinary agents of infection, the pneumococcus and still more unusual forms of bacteria may be met with—streptothrix, actinomycosis, the typhoid bacillus (McClintock), etc.

Even when an abscess is directly due to a chronic suppuration of the middle-ear or frontal sinuses it may have no apparent connection with the original site of disease; in other cases there may be a local or general infection of the meninges. This may occur secondary to rupture of the abscess itself into the subarchnoid space—an incident which is particularly apt to follow a misdirected operation. Lossen has shown that the cerebellum may become infected by extension of suppuration along the sheath of the acoustic; also that cerebellar abscesses are particularly apt to be complicated by sinus thrombosis, meningeal abscess, or meningitis.

Symptoms.—When secondary to a chronic otitis media—and cases of this sort may be taken as typical of all—there appear in sequence certain new symptoms not previously observed. For example:

A patient 30 years of age had had, since an attack of scarlet fever in childhood, a chronic discharge from the right ear. He had had several acute exacerbations of the local disease, none of them serious until the present flare-up of the trouble. Two weeks before his admission there had been an increase in the discharge and some granulations were removed from the meatus. Two days later he complained of some headache and nausea, with fever and chilly sensations (*initial stage*). During the following 10 days these symptoms abated somewhat, but did not entirely disappear (*latent stage*). Then, with a sudden cessation of the discharge from the ear, there followed a severe and constant headache, vomiting, a pronounced change in his mental activities, with slowness of thought, drowsiness, irritability, and defective memory. The pulse was slow, his temperature became subnormal, and there was a leukocytosis of 22,000 (*manifest stage*). There were no focal symptoms. Percussion over the temporal bone on the side of the disease elicited marked tenderness. An abscess was found in the temporal lobe, with no evidence of meningeal involvement.

This may be taken as a fairly typical instance of the clinical picture, showing the stages of the disease in their regular succession. When death occurs (*terminal stage*) it is accompanied by high temperature and the usual compression phenomena with respiratory paralysis.

The clinical history is of paramount importance in these cases, for there are no absolutely diagnostic signs. Other than the symptoms thus recorded (the cessation of discharge; the headache, often severe, rarely absent; vomiting; perhaps a chill; often, though not always, a subnormal temperature and slow pulse; drowsiness; irritability; slow cerebration; delirium and loss of memory; tenderness to percussion, etc.), may be mentioned the following: Facial paralysis may occur and be homolateral from involvement of the peripheral nerve itself, or contralateral, together with evidence of compression of the lower precentral area, with aphasia. Occasionally there is contraction of one pupil, followed later by dilatation. A choked disk may develop. Mental symptoms are especially pronounced in frontal lobe abscesses. Change of position is apt to lead to dizziness.

If the acute symptoms subside, if encapsulation take place, and if the abscess be situated in a "silent" region, it may remain dormant for years and only be disclosed by accident at a post-mortem examination. Occasionally when the lesion is situated in the frontal lobe and has led to mental derangement, the victims may become inmates of an asylum and be regarded as chronically insane.

Even in the presence of suspicious symptoms the seat of the lesion is often most difficult to determine. The best guide, on the whole, is the situation of the external process which presumably has led to it. In abscesses of the temporal lobe, unless they have involved the speech

mechanism or the lower motor centers, there may be no localizing symptoms. Abscesses of the cerebellum are particularly difficult to recognize, although suboccipital pain, vertigo, dizziness, nystagmus, or cerebellar ataxia may be suggestive. v. Beck regards a stiff neck and turning of the head toward the side of the disease as important. Cerebellar "seizures," which consist of sudden, unexpected attacks of vertigo, roaring in the head, relaxation of the limbs, and falling to the ground in a semiconscious condition (Dana), may be helpful.

Diagnosis.—In the acute cases one must distinguish between abscess, encephalitis, meningitis, ependymitis, and septic sinus thrombosis. When there is no clear sequence of events such as have been described, a certain diagnosis may be impossible. The chronic cases are not infrequently mistaken for tumors, since they may present the same underlying pressure phenomena.

The history is often of chief importance, for a neurological examination, particularly in the absence of focalizing symptoms, is most unsatisfactory in a patient sufficiently ill to lead to a suspicion of abscess. There are certain points which are helpful in differentiation. The evidences of brain pressure, the stupor, the slow pulse, respiratory changes, choked disk of a low grade, and a subnormal temperature, are apt to be more marked in abscess than in meningitis or encephalitis, except in their late stages. Cervical rigidity and Kernig's sign are more characteristic of meningitis than abscess, and the former usually has a higher leukocyte count (Starr.) Sinus thrombosis has a characteristically irregular fever; there is tenderness at the tip of the mastoid and the jugular; exophthalmos follows when the cavernous sinus is involved. There is no history of suppuration in the cases of acute hydrocephalus following ependymal inflammations, and here choked disk appears early and reaches a high grade.

The differentiation between abscess and meningitis may at times be made by an examination of the fluid obtained by a lumbar puncture. The cerebrospinal fluid, even if bacteria are absent, shows an increased number of leukocytes, both in tuberculous and in the early stages of ordinary septic meningitis. This is not true of abscess, unless it be complicated by meningitis. Allan Starr regards this as a valuable aid. Fuchs and Rosenthal have found as high as 952 cells in tuberculous meningitis and 100 in general meningitis; whereas there should normally be but one or two in a centrifugated specimen of 5 cc. Neumann and Grunert found no bacteria in the cerebrospinal fluid. Indeed, in many abscesses of long standing cultures from the abscess itself prove sterile. There is, I think, always a certain element of danger in a lumbar puncture, since the altered conditions of tension may lead to a rupture of the abscess, particularly if it be cerebellar; only a small amount of fluid, therefore, should be withdrawn.

There are two complications of otitis media which I have found to offer especial difficulties of diagnosis. In one of them symptoms closely resembling intracranial extension of disease may be produced by a reflex disturbance through the trigeminal nerve. These patients show no rise in temperature; but great tenderness, headache, nausea, and even vomiting may occur. The tenderness, however, is superficial and conforms with the trigeminal sensory skin field; even the hair can not be touched without discomfort. I have seen several cases of this sort recover without operation, and they may possibly represent a mild degree of dural involvement. The other complication, due to an acute serous meningitis (not, so far as is known, of bacterial origin) is still more common and may closely simulate abscess. In these cases a lumbar puncture or a decompressive craniectomy with evacuation of the fluid proves curative; should, therefore, a superabundance of clear fluid be disclosed in an exploratory operation for abscess it is wise not to be too persistent in the search for a pocket of pus.

The *prognosis* without operation is absolutely bad; with operation it is not brilliant. Even in experienced hands the mortality remains about 50 per cent; for an abscess may not be found, and if it is found and evacuated there may arise unavoidable and often fatal complications—a second abscess due to inefficient drainage, a fungus cerebri, purulent meningitis, septic sinus thrombosis, pyemia, etc.

Treatment.—Most important of all therapeutic measures is prophylaxis. The fact that cerebral abscess is less commonly observed than formerly may be accounted for by the greater promptitude and greater skill shown in the treatment of those diseases whose neglect leads to it. Owing to Macewen's book, which has had a deserved popularity among practitioners in general, rhinologists and otologists are alive to the fact that early radical treatment of suppurating processes, while they remain extracranial, is the best treatment for these intracranial complications. Probably more than one-half of all cases are secondary to suppuration in the otitic and rhinitic cavities, and if they become involved during the course of such infectious diseases as pneumonia, influenza, scarlet fever, typhoid, etc., when patients are seriously ill and unable to call attention to local discomforts the infection may easily be overlooked.

When an abscess has formed in spite of all care, the condition is serious; a critical operation must be resorted to; and even with our modern methods the mortality remains high. Up to 1884 only 55 cases of operation had been reported (Körner) and Bergmann, in 1889, could find a record of only 8 recoveries. Since Macewen's treatise (1893), however, the operations have so multiplied that they cease to be regarded as rarities. By 1898 there were records of 60 successful operations upon temporal and 12 upon cerebellar abscesses

(Marsch). Unfortunately, however, these operations still continue to be conducted as a last resort in the "manifest" or even near the "terminal" stage of the disease. They should, on the other hand, be undertaken early, without waiting for unequivocal symptoms, for, as Körner says, "Auf mehr Zeichen warten, heisst auf mehr Leichen warten."

Method of procedure.—It is Macewen's opinion that in the otitic cases it is wise, when there is a question of cerebral abscess, to open first of all through the squamous portion of the temporal bone. Otologists, as a rule, owing to their greater familiarity with the method of approaching the dura through the mastoid region and then through the tegmen tympani, favors this posterior route. There is much to be said for both views. The surgeon who is particularly familiar with cranial operations, in case there is some doubt as to the nature of the intracranial complication, might prefer, as a primary measure, to open the skull through the squamous portion of the temporal bone, in order to expose the dura and, if necessary, the temporal lobe in the middle cranial fossa; for should the exploratory intracranial operation be conducted through the diseased ear, the risk of producing infection would be greatly enhanced. On the other hand, the otologist, after cleaning out the mastoid cells, the infected antrum, and middle ear, is inclined to wait for a day or two to see whether the suspicious intracranial symptoms do not subside.

I am not convinced which of these methods is the better, and have followed at times one, at times the other: judgment must depend upon the individual case. With experience it is a simple matter to explore intracranially the roof of the petrous bone, the dura overlying it, and, if necessary, the temporal lobe without risk. I prefer to do this by the muscle-splitting operation. In case nothing is found this wound may then be closed—a measure which would perhaps be unsafe if the infected field had been primarily opened up. Particularly in the "initial" or "latent" stages, when diagnosis may be in doubt, the primary mastoid operation is advisable, leaving an open wound. If the suspicious symptoms should continue the opening may be so enlarged and deepened as to remove a part of the tegmen tympani; possibly an extradural focus may be disclosed, or if not, the temporal lobe may be explored with a hollow needle from below. Many successful operations performed by this route have been reported, most of them by otologists. When there is a question of involvement of the cerebellum I think there is no doubt but that this latter method is preferable; namely, to clean out the mastoid cells, antrum, and ear first, and at the same time to investigate the sigmoid sinus. Unless symptoms are urgent the cerebellar exploration may be deferred for a day or two.

When an abscess has been found it should be treated like an abscess elsewhere, with free incision and drainage. It is, however, often difficult to accomplish this, owing to the fact that about the edges of the incision in the brain the cerebral substance becomes edematous, swells, and tends to occlude the opening and retain the discharges. Furthermore, unless meningeal adhesions have formed—and they are unusual—there is a likelihood of meningeal infection as a result of cerebrospinal fluid leakage alongside of the draining abscess. While the brain harbors an abscess it is tense and the exposed cortex tends to protrude through the opening which has been made in the dura; after the abscess has been evacuated this tension subsides and the cerebrospinal space once more opens up and can be reached by the infection.

When, however, adhesions are present and serve to bind together the dura, arachnoid, pia, and cortex, the approach to the abscess may be conducted with a minimum of risk. Owing to the fact that an abscess may assume a mushroomlike shape with a narrow stalk at the original site of infection, it may often be opened through this stalk without doing further damage to the cortex than has already occurred from the disease (Ballance). Preysing's figures well illustrate this type of abscess, and such conditions give a particularly favorable operative prognosis.

In exploring for an abscess it is essential to use a proper hollow needle with a blunt end and with one or more openings, which should be on the side. The brain should be freely incised on a director introduced to the same point at which the pocket of pus may chance to have been entered. It is well to avoid irrigation of an abscess, though this is a procedure commonly followed. Free drainage is the essential thing, and in the absence of further symptoms the gauze should remain long undisturbed. The patient should lie with the opening downward.

A complication which may arise during the operation, especially when conducted in the "manifest stage" of the disease, is respiratory failure from the additional burden of the anesthetic on an already embarrassed respiratory center. It is essential in these operations, therefore, that an artificial respiration apparatus be at hand, for when put in use it is possible to continue the operation, and if an abscess should be found and evacuated the spontaneous resumption of respiration with recovery is possible. Many remarkable cases of this primary respiratory failure with continuance of cardiac activity have been reported. I have had one patient in whom, though the operation was completed under artificial respiration and an abscess was found, there was no resumption of spontaneous breathing, though the heart continued to beat for 23 hours; on another occasion a patient was saved.

CONCUSSION, CONTUSION, AND COMPRESSION.

These terms, of time-honored usage, are often loosely employed and with but faint understanding, not only of the pathological lesions underlying the conditions so designated, but even less of the physiological phenomena they evoke. They deserve, nevertheless, to be retained and, as is the custom, they will be considered together.

The three conditions shade so imperceptibly into one another that it is often difficult to determine where true concussion ends and symptoms of contusion begin; and likewise where symptoms of contusion end and those of compression begin. Indeed, in the minds of many, concussion is but a grade of contusion and, furthermore, cerebral contusion is impossible without some degree of compression, whether from hemorrhage or edema, provided the skull is closed and the cranial bones remain intact. A single illustration will serve to show how these states may overlap:

A workingman fell from a scaffold on his head. "Concussion" was evidenced by the immediate loss of consciousness. His failure to recover from this state in the course of a few hours raised a suspicion of "contusion" and free blood would have been found in the arachnoid space. He died in a short time with symptoms of "compression." At autopsy there was found a laceration of the orbital surface of the right frontal lobe with a large extravasation which had forced its way into the brain substance alongside of the basal ganglia.

Concussion and contusion are the effects solely of traumatism. The symptoms of compression likewise follow an injury when, as in the case just cited, it leads to intracranial hemorrhage or to swelling of the brain within the closed skull. In its varying grades, however, compression is even more often the result (1) of spontaneous hemorrhage, as in apoplexy; or (2) of edema originating in other than traumatic sources, such as nephritis, or disturbed circulatory conditions of the cerebrospinal fluid the result of ventricular hydrops, or meningeal inflammation; or (3) of the foreign-body effects of an abscess or new growth within the cranial chamber.

Concussion (*Commotio cerebri*; *hirnerschütterung*, etc.).—An individual may be momentarily stunned by a slight blow upon the head. No subsequent ill effects need follow. A more serious blow may lead to an actual temporary loss of consciousness, on recovery from which certain sensations of weakness, dizziness, or headache may persist for a time. A still more violent blow may produce unconsciousness, even coma, of longer duration, and may leave the victim mentally disturbed for hours or days, with loss of memory of the events connected with the injury; with headache, possibly nausea and vomiting; with ataxia and dizziness, especially when an upright posture has been assumed; and perhaps even with medullary symptoms, shown by slow pulse or respiratory changes. All of these symptoms

may endure for days and, in fact, often may leave the patient a permanent victim of post-traumatic neuroses.

Morbid anatomy.—To what are these symptoms due? Though experimentalists and pathologists have long endeavored to answer this question, they have not done so to the satisfaction of all. Patients in whom serious conditions of concussion have led to death may show at autopsy no cerebral lesion whatsoever. They may at other times show certain foci of extravasation scattered throughout the brain—in other words, microscopic evidences of contusion. These lesions and the symptoms which they produce bear no definite relation to any cranial injury. The skull may or may not have been broken at the time of the injury and, indeed, some of the most serious cases of concussion are unaccompanied by fracture. It is true that most injuries which suffice to cause a fracture are accompanied by symptoms of concussion or contusion, though this is not invariable. Local depressed fractures, for example, can occur without even a momentary loss of consciousness, this being the one cardinal feature of concussion.

The view is held by some that a simple jar of the brain may so disturb the molecular integrity of the nerve cells as to lead to unconsciousness, even to coma and death, and yet produce no lesion which is recognizable, even by a most thorough histological study. As shown by Koch and Filehne, and later by Witkowski, symptoms which are comparable to those of concussion as it is seen in man, may be elicited by rapidly repeated blows upon the cranium of an animal—unconsciousness, slow pulse, relaxation of the muscles, insensibility to sensory stimulation, etc. Even should these symptoms be severe enough to result in a fatality, a post-mortem examination may show nothing more than some evidence by hyperemia of the brain and meninges. Fischer believed that the symptoms are occasioned by a reflex paralysis of the vasomotor center, which leads to a sudden failure in blood pressure analogous to that which occurs in shock.

On the other hand, patients in whom symptoms of concussion have not been severe may die from the effects of concomitant injuries, and, contrary to expectation, the brain may be found at autopsy more or less lacerated and containing scattered foci of extravasation due to capillary hemorrhages. When such extravasations occur in the neighborhood of the pons or medulla they are commonly fatal. Such findings are naturally classified as contusion.

Upon these different views two schools have grown up: (1) Those believing, with the late von Bergmann, that simple concussion may exist without extravasation, and (2) those agreeing with Kocher in regarding concussion as the result of minute contusions scattered

throughout the brain in consequence of the transmitted effects of the blow.

Symptoms.—Disturbances of consciousness are essential to concussion. The victim may be in a simple lethargic state from which he can be aroused, or in the deep sleep of a drunken person. As unconsciousness passes away general evidences of cerebral disturbance appear. There is headache, vertigo, often nausea and vomiting, possibly convulsions, often a subnormal temperature, at time changes in the cardiac and respiratory rates, and almost always more or less loss of memory of the events incidental to the injury. In addition to these general symptoms, inasmuch as many cases of simple concussion are complicated by symptoms due to laceration of the brain and extravasation of blood, focal symptoms may be present.

As indicated in the introductory paragraphs, the severity and the duration of the symptoms of concussion are most variable. In a mild case recovery occurs after a brief interval. In more severe cases, when consciousness has been restored, the patient may perform more or less automatic acts of which he subsequently has little memory, and he may be left for a time incapacitated, both physically and mentally. In still more severe cases profound unconsciousness may endure for a considerable time, during which the reaction to external stimuli is abolished, such as failure of the conjunctival reflexes. The pupils in the early stages are apt to be contracted. There may be involuntary passage of urine and feces. If this condition persists for some hours or days it is probable that, in addition to the concussion, more serious lesions have been produced.

In some instances of concussion a period of excitation may follow the period of depression of the vital functions. The patient may become violent, irritational, and may have to be forcibly confined. An early rise in temperature has been observed in simple cases of concussion. In severe cases it is not unusual for the ten days or two weeks of convalescence to be attended by a subnormal temperature and slowed pulse.

Prognosis.—Any case of cranial injury which results in concussion must be given a guarded prognosis, not only as to the immediate outcome, but also as to the final restoration of normal cerebral activity. Owing to edema alone, symptoms of compression may supervene in what might have been considered a simple case of concussion; convalescence may be indefinitely protracted, owing to the headache, mental depression, etc., which characterize the familiar post-traumatic neuroses that oftentimes follow such injuries.

Treatment.—A careful examination of the head should be made in view of a possible fracture. In questionable cases it is wise to shave the scalp. Should there be evidence of failure in arterial tension, due to the temporary upsetting of the vasomotor center, efforts

should be made to improve the circulation by cardiac stimulants or, better, by measures directed toward supporting the relaxed peripheral vessels, particularly those of the splanchnic field, as by bandaging the extremities or by pressure on the abdomen. The patient should be kept quiet, warm, and his head low. An ice cap should be applied. Atropin is said to be a desirable drug.

In case the early symptoms have been severe, it is essential that enforced rest and quiet should be continued for a period of from 10 days to two weeks, with daily free evacuation of the bowels, an ice cap, and a light diet. Should a suspicion of further complications arise and especially should pressure symptoms supervene at any time, a decompressive operation is indicated. It may be wise to precede this by a lumbar puncture, which will indicate whether there has been an extravasation of blood or whether the symptoms are simply due to an excess of fluid from edema. In the latter case the puncture alone may often serve to alleviate them.

Contusion.—Concussion, as has been said, may be regarded as nothing more than a low form of contusion, even though there may be no demonstrable evidence of bruising. In case these contusions result in extravasation, macroscopical evidences of hemorrhage may be present, and from these small foci of extravasation we may pass through all grades of contusion up to actual laceration of areas of the brain. Post-mortem examinations after traumatic death occasionally reveal unsuspected lacerations of wide extent, even when symptoms of concussion have been relatively slight. Cases of this sort argue somewhat in favor of a clinical differentiation between concussion and contusion.

Contusions with or without laceration, as pointed out by Duret, are most apt to occur at the tips of the temporal lobe and base of the frontal lobes. These lacerations are a frequent accompaniment of basal fractures, for the same injury which suffices to produce the laceration is likely, at the same time, to cause a bursting fracture of the skull. Contusions often occur at that part of the brain opposite to the point of external traumatic impact, and consequently these cerebral lesions afford a better illustration of the effects of injury by *contrecoup* than do those of the skull itself.

Course.—What has been said of concussion applies likewise to the symptomatology, diagnosis, and prognosis of contusions and lacerations of the brain, though in them the immediate symptoms are usually much more pronounced. The period of "concussion" is longer and certain evidences of compression almost always supervene: early when due to hemorrhage, later when brought about by contusion edema.

Extravasations from the injured vessels vary greatly in number, in situation, and in extent. They may be intracerebral and multiple,

and in case they involve important centers may be rapidly fatal without giving symptoms of compression.

Most forms of laceration, however, primarily affect the cortex, particularly at the base; consequently the presence of blood in the subarachnoid space is almost invariable, and as this can easily be determined by a lumbar puncture, this measure should become, for diagnostic purposes, a routine procedure in all suspicious cases. At times the extravasation which follows even a small area of contusion may be large and intracerebral, leading to death from compression.

Not only are the symptoms of contusion more pronounced and more enduring than are those of simple concussion, but convalescence as well is more protracted and patients are almost certain to be troubled for some time with symptoms of headache, nervousness, irritability, and mental depression.

Treatment.—As in concussion, enforced rest is essential and should be persisted in for at least a period of two or three weeks. These patients almost invariably reach a certain stage of well-being in which they wish to escape from confinement and resume their work long before they are fit to do so. They are almost always discharged from hospital care earlier than the nature of the lesion justifies. Until there is a complete cessation of headache; until the pulse rate, which is almost always slowed after such a lesion, reaches the normal; until there is a complete disappearance of evidence of intracranial pressure shown by some stasis in the eye grounds, they must be kept quiet, on a light diet, and recumbent.

Many of the more severe symptoms accompanying contusion may be avoided by properly conducted surgical procedures. Lacerations of the temporal lobe which have led to local intermeningeal extravasations may be greatly benefited by operative measures. When there is doubt about the condition an exploration through the split temporal muscle, with removal of bone, will determine whether or not there is any lesion present; and often the period of post-traumatic edema may be shortened and the symptoms which accompany it lessened by such a measure. It is preferable needlessly to operate on some borderline cases than to neglect those which might thus be benefited.

Compression.—With the exception of a small amount of cerebrospinal fluid, the brain, including, of course, its meshwork of blood vessels, normally fills the cranial chamber. The cerebral nervous tissue itself is as incompressible as water. In so far, however, as the brain is a vascular organ, it may be made smaller by having some of its blood supply squeezed out through pressure. Thus a foreign body experimentally introduced into the cranial chamber makes room for itself by a local emptying of vessels in the neighborhood, leading to the so-called *local increase in intracranial tension*. The compression

effects of such a local disturbance diminish with the distance from the primary seat of pressure, and only in case they are of high degree are they felt throughout the entire chamber. On the other hand, should fluid under pressure be let into the cranium in such a way that it distributes itself in the subdural space through the entire chamber, leading to an equable pressure or tension in all parts, we may speak of the condition as one of *general increase in intracranial tension*. Compression which results from processes of these two sorts must be differentiated. No form of encroachment, however, on the intracranial space fails to affect the cerebral circulation, and it is this circulatory disturbance which plays the chief rôle in eliciting the phenomena of compression.

There are exact clinical counterparts of these experimentally induced conditions. Thus, a tumor or abscess, a focal edema, a vascular extravasation, whether it be outside the dura or in the brain substance itself, an operation such as the Gasserian ganglion operation which necessitates elevation and compression of a lobe of the brain, etc.—any one of these things may lead to a local increase of tension, the pressure effects of which diminish in proportion to the distance from the local process. On the other hand, in meningitis, in hydrocephalus, in general edema, in widespread basal hemorrhages, or in the congestion secondary to extensive sinus thrombosis, the increase of tension is more general and its effects are exerted in equal degree against all parts of the cerebrum.

Compression, therefore, may occur in a variety of ways; either through the accession of some abnormal substance, or through an abnormal accumulation of some substance already present. Thus, a new growth or an abscess may produce compression, provided the space, which the brain normally should occupy, is encroached upon; either of them may, however, be present and elicit no symptoms of compression in case they destroy the brain as they progress. Again, an increase in the normal amount of cerebrospinal fluid may lead to symptoms of compression in case the fluid is more rapidly formed than usual or, as in obstructive hydrocephalus, in case it finds unusual difficulty in escaping from the cranial chamber. Edema, the result of injuries or of chemical disturbances in the body fluids brought about by nephritis, may likewise be the source of compression.

The symptoms, however, in many of these conditions, owing to the fact that the cerebral lesions progress slowly, are not exactly comparable with the symptoms which have been produced experimentally; for experimental studies have been devoted largely to the consideration of those phenomena which follow an acute increase of intracranial tension. Consequently the laboratory observations are more directly comparable with clinical cases of acute compression,

such as occur in hemorrhage, rather than with cases of slowly progressive increase in tension, due, for example, to tumor.

Physiological effects of acute compression.—Galen, Boerhaave, Haller, Magendie, and many others recorded observations on the symptomatic response to pressure against the brain. In recent time, more complete studies have been made by Leyden, Pagenstecher and Duret, by Bergmann, Naunyn and Schreiber, by Spencer and Horsley, Leonard Hill, Kocher, the writer, and many others. A few of the more important factors which have been brought out in the laboratory may be mentioned, in order that we may better understand the physiological responses to compression which we encounter in clinical cases.

It has been emphasized by some that the first effect of any encroachment on the intracranial space is to drive out of the chamber the small amount of cerebrospinal fluid which is normally present. A further encroachment can only occur by crowding out of the blood-vessels a certain amount of their content, and inasmuch as the tension in the cerebral veins is lower than in the other vascular branches, these vessels are the first to suffer. Venous stasis, therefore, is one of the first consequences of the pressure. A further augmentation of the compressing force, with still greater encroachment on the intracranial space, soon brings the pressure exerted against the brain up to the level of the pressure in the capillaries and even to the arterial tension, and, needless to say, cerebral anemia results, with early and irrecoverable loss of function in those parts of the brain whose circulation has thus been completely shut off for any considerable time.

We must continually bear in mind the difference between a local and a general increase in tension. Inasmuch as the pressure effects of a local process are greater in its immediate neighborhood than at a distance, and inasmuch also as there is considerable pressure discontinuity between the three intracranial compartments, owing to the fairly rigid partitions formed by the falx and tentorium cerebelli, a local pressure, let us say over one hemisphere, may exceed the local arterial pressure and lead to a local anemia sufficient to throw the adjoining parts of the brain out of function without seriously affecting the other hemisphere or the subtentorial structures, of which the medulla is of prime importance. On the other hand, if a compression of like degree had been distributed equally throughout the cranial chamber by means of fluid let into the subdural space under pressure, a generalized anemia would have followed and death would have ensued from implication of the vital centers in the medulla. In a similar way we may produce general compression and elicit the symptoms in their successive stages by pressure on the sac of a cephalo or meningomyelocele.

As the medullary centers are the crux of the situation, it can be readily inferred that an acute local compression in the neighborhood of the medulla is just as serious as a general compression of like degree, whereas over the hemispheres a much higher grade of local compression is possible without producing death. Thus it is that an arterial hemorrhage (apoplexy) may occur in one of the hemispheres and lead to an absolute local anemia, since the pressure of the extravasation is equal to arterial pressure without producing a corresponding degree of anemia of the medulla, otherwise all of these cases would of necessity be immediately fatal.

It has been stated that when subtentorial tension—in other words, the pressure against the medulla—reaches the arterial pressure, death must ensue in consequence of anemia of the vital centers. Certain reservations must be made to this statement, for if it implies *normal* arterial pressure it is erroneous. For when the external pressure against the medulla begins to approach or equal the arterial tension, the anemia stimulates the vasomotor center, the general arterial pressure is raised, and the medullary centers are again sufficiently well supplied with oxygenated blood. If the external pressure is still further raised, the same response on the part of the vasomotor center again occurs, and thus in progressive stages the arterial tension may be raised higher and higher until, at times, it may reach more than twice its normal level.

This progressive rise in tension of the circulating arterial blood, due, as has been shown, to constriction of the splanchnic field, often-times does not permanently continue on the exact level to which it has been forced—namely, slightly above that of the intracranial tension. On the contrary, it often fluctuates above and below this level, with a definite periodicity (exaggerated Traube-Hering waves), and hence it is that rhythmic respiration of the Cheyne-Stokes type is produced, for during the period of fall anemia results and the respiration fails; when the arterial pressure rises again the medulla is resupplied with blood and respiration is resumed. This reaction may continue for hours and has been particularly well described in Eyster's papers.

Finally, a time comes when the regulatory mechanism is no longer efficient and then, whether from a further increase in external pressure or from fatigue of the vasomotor activity, the arterial pressure drops permanently below the level of the pressure exerted against the medulla; anemia results, the respiratory center fails, and the heart keeps on beating as an isolated organ, uncontrolled by vagus or vasomotor activity, until, after a variable time, with fall in pressure to zero, it ceases altogether through asphyxiation.

This brief review of the phenomena, which have been experimentally observed, may suffice to show, as Leonard Hill has emphasized,

that compression symptoms are not due to mechanical excitation or structural injury, but, on the contrary, to circulatory disturbances—a primary venous stasis, resulting finally in capillary anemia. It serves also to point out the differences between a local and a general increase in tension: it shows that anemia of the medulla plays the chief rôle in eliciting the so-called major or bulbar symptoms of compression—namely, the high blood pressure from stimulation of the vasomotor center, the slowed pulse from vagal stimulation, and the rythmic respiration of the Cheyne-Stokes type which hinges on the fluctuation in level of the raised arterial tension, which, for a short period, leaves the respiratory center anemic and then resupplies it with activating blood.

Symptoms.—We have seen that compression of the brain can only take place at the expense of emptying certain of the blood vessels, and, furthermore, depending upon the character of the process through which the pressure is exerted, that the interference with the circulation may, on the one hand, be confined to a more or less restricted field or may, on the other, be generalized over the entire cerebrum.

In view of the gradations of circulatory disturbance, as well as of the symptoms they occasion experimentally, Kocher, in his comprehensive monograph, has endeavored to subdivide the progressive phenomena of compression into four stages; and for purposes of clinical designation it is convenient to have such a classification of cases or of stages of the individual case.

His first stage (*Compensationsstadium*) corresponds with a mild grade of compression, or with the early stage of what may become a severe grade, in which the pressure exerted against the brain by the foreign substance is not sufficient to seriously compromise the circulation. By the escape of cerebrospinal fluid and by a narrowing of the venous channels the process is accommodated with nothing more than a certain degree of venous congestion, which may be local. Symptoms are in the main insignificant, particularly if the process is remote from the medulla. There may be some headache, possibly certain focal symptoms referable to the site of the lesion, some mental dullness, and little else. There is but slight interference with the circulation of the brain as a whole.

His second stage (*Anfangsstadium des manifesten Hirndruckes*) corresponds with the beginning failure of circulatory compensation. There is sufficient venous stasis to lessen the normal amount of blood flowing through a considerable part of the capillary field. Headache is more pronounced and there may be vertigo, restlessness, a disturbed sensorium with excitement or delirium, an unnatural sleep, etc. Other and objective symptoms become manifest, particularly shown as a slight venous stasis of the extracranial vessels. The face

appears somewhat cyanotic, the venules of the eyelids are dilated, and of greatest import, a distention and tortuosity of the veins radiating toward the optic papilla, with or without evidence of beginning edema of the nerve head, may be found on an ophthalmoscopic examination. Indications that the venous congestion is affecting the medullary circulation is shown by a slowed pulse and possibly by a slight rise in blood pressure.

His third stage (*Höchstadium des manifesten Hirndruckes*) corresponds with the stage of widespread capillary anemia brought about by further increase of the tension. Here the medulla will not have escaped even though the lesion causing the pressure is a focal one and lies remote from the hind brain. The period of vasomotor regulation has set in with its high blood pressure, and this, combined with its vagal quality, gives to the pulse its so-called "bounding" character. The rise in arterial pressure may exhibit fluctuations in level, which can easily be recorded on a sphygmomanometer, even when they are not appreciable to the finger. These are accompanied by rhythmicities in respiration, which may acquire the typical Cheyne-Stokes type with periods of absolute apnea; by rhythmic alterations also in the size of the pupils; by a wavering increase and lessening of the depth of stupor, so that with the "up wave" in pressure, the patient may moan, become irritable, and thrash about, with the "down wave" be deeply comatose; and by other signs indicative of the vasomotor rhythm. The pulse is slowed even to 40 or 50 beats per minute. The choking of the optic disks is more pronounced. The reflexes may become abolished. Cyanosis of the face is extreme, the respiration snoring, and the patient approaches the brink of failure of the regulatory vasomotor responses.

In the fourth or terminal stage (*Lähmungsstadium*) compensation on the part of the arterial tension shows signs of failure; there are irregular cardiac and respiratory efforts, the pulse grows rapid, coma deepens, there is complete muscular relaxation, the pupils become widely dilated, and with the permanent fall in blood pressure there is a cessation of all cerebral function with respiratory paralysis.

Prognosis.—States of compression not only vary so greatly in degree, but also may be due to such a variety of lesions that it is impossible to consider fully the course and prognosis of the condition, even as it appears in any single malady. The pressure effects of a tumor or of chronic hydrocephalus, for example, differ materially from those of the acute lesions that have been described. Possibly because they represent asthenic states they do not exhibit the same pronounced responses on the part of the vasomotor center that characterize other and sthenic conditions. The slow progress of the disease may allow of a circulatory adjustment not possible in the more acute lesions, and although a sudden death from compression some-

times befalls these patients, it is apt to be due to paralysis of vital centers by invasion or by a suddenly forming local edema.

In the acute cases, those with hemorrhage for example, the prognosis hinges on the size of the compressing mass, the rapidity of its formation, the length of time which has elapsed, and the stage of compression which is reached. A patient in the third stage, with high blood pressure, and especially when respiratory difficulties with snoring or rhythmic alterations are in evidence, must be regarded as in a most precarious condition. For, even though the pressure may not advance and though the vasomotor mechanism suffices to tide over the threatened medullary anemia, there is always a chance that an inhalation pneumonia may follow. The immediate prognosis for life in all cases which do not reach the "high stage of manifest compression" is good, although the extent of damage from the local lesion, whatever it may be, may suffice to leave incapacitating paralyses. This, however, is another story, to be taken up with the consideration of the various lesions which produce compression.

Treatment.—Only certain general rules can be laid down; the more specific ones must be deferred for other sections. Needless to say, we must meet with mechanical measures a condition—viz, pressure—which is produced by mechanical forces. This means, on the one hand, a removal of the source of pressure, be it an accessible clot or abscess, a tumor or increase of cerebrospinal fluid; on the other hand, a release from the effects of the lesion by an opening in the skull—a decompression, in other words—in case the lesion is inaccessible or is irremovable, as in edema, etc. The surgical methods of accomplishing these results will be considered elsewhere.

There are two other measures seemingly less radical than a cranial operation, both of which have their advocates, both of which possess elements of danger—lumbar puncture, with the idea of making room by removing fluid, and venesection, with the purpose of lowering the high blood pressure and of "depleting" the brain. In cases of compression of high grade a lumbar puncture may be promptly fatal, owing presumably to the fact that the intracranial pressure wedges the medulla and lips of the cerebellum into the foramen magnum when the support of the spinal fluid has been removed. Numerous sudden fatalities have been recorded as a result of this measure.

Venesection, particularly in cases of compression from hemorrhage, has been considered advisable on the view that the high arterial pressure is the cause of the hemorrhage and is likely to increase the bleeding, rather than that it represents a conservative process on the part of the organism to overcome cerebral anemia. Falkenheim and Naunyn recommend that the blood pressure should be supported by every means in these cases. Bergmann and Hill, on the other hand, are inclined to the traditional treatment of lowering it by blood-

letting. It has been my experience, both in the laboratory and clinic, to see disastrous results from venesection with lowering of blood pressure in cases of compression in its third or highest stage. Only in cases which rest in the earlier stages, with an abundant reserve on the part of the vasomotor center, can blood be withdrawn with impunity, and in these cases there is rarely any symptomatic indication for bleeding.

3. **The psychoses and insanity following cranial injuries.**—Aberrant mental processes are a common result of injuries to the head. We may consider (1) their immediate and (2) their late manifestations.

(1) *The immediate disturbances.*—In reviewing the symptoms of concussion and contusion some of the acute symptoms have been recorded. After the usual initial period of unconsciousness the patient remains stunned or dazed for a variable period. He suffers from headache; becomes dizzy; is somewhat nauseated on change of position; is often in a dreamy condition of sopor, from which he can be aroused for a few moments with an apparent feeble grasp of his situation, but into which he soon lapses again. He may pass from this dull, stuporous state into an active one, accompanied by violence, delusions, disorientation, and delirium. This condition may become protracted over a period of a few weeks and represents a *primary traumatic insanity*. There is often fever. Forcible restraint of these patients is at times necessary.

Complete recovery after these symptoms with a defective memory of the events connected with them is not unlikely, even in the extreme cases; and it is quite probable that they are often due to states of acute edema. I have seen patients in whom compression symptoms, even to the point of medullary paralysis, were present, and in whom a decompressive operation disclosed an abundance of sterile fluid under pressure (acute serous meningitis).

(2) *The late or residual disturbances.*—These occur in a group of cases which make a satisfactory and early recovery from the injury, but show after-effects—the so-called “posttraumatic neuroses.” These patients complain of sensations of pain on pressure or paresthesia of one sort or another, often referred to the seat of the primary external injury. They have attacks of dizziness, irritability, and nervousness. Their traits may change; formerly good natured, they become brooding, moody, introspective, irascible, and even violent at times. They are forgetful, make stupid blunders, are slow of thought, have no power of concentration, and hence their capacity for continuing their former occupation is greatly lessened. There is often excessive sensitiveness of the head to jars or noises, and a stooping posture is not tolerated. Natural sleep is affected. They are most intolerant of alcohol or tobacco in amounts previously taken without ill effect. They are very susceptible to febrile disorders.

Though no objective signs accompany these complaints, they are so uniform from case to case that the symptoms can not be regarded as other than genuine. They are a frequent source of litigation. Epilepsy not uncommonly occurs and attempted suicide is not unusual. These patients often clearly develop a *secondary traumatic insanity*.

There seems to be no definite relation between any special types of lesion and the particular form of psychosis which may follow. Simple concussion or contusion, with or without definite fracture, fractures of the base or of the convexity, cranial injuries leading to loss of substance or otherwise, gunshot injuries, and other forms of lesion may at times provoke these symptoms in varying grades. No more does there seem to be any definite relation between the situation of the cerebral lesion and the occurrence of these symptoms, though one would suppose that injuries of the frontal lobe would be most likely to lead to them. Doubtless a constitutional peculiarity, inherited weakness, or alcoholism predisposes to post-traumatic psychoses.

No surgeon fails to see a number of these unfortunates. They appear again and again in the same hospital wards, or more often wander from clinic to clinic in the vain hope of securing some surgical relief for their miseries. Many of them, particularly when cranial defects or local scars are present, are subjected to operation and reoperation at various hands. They are received with scant welcome, and when weary of their complaint the ward discharges them "untreated," with a diagnosis of traumatic neurasthenia or neurosis. They finally wear out the patience and sympathy even of their home people. Little wonder that there is often an attempt, sometimes successful, at self-destruction.

Treatment.—As is the case with many of the sequels of cranial injury, the treatment is largely preventive. We have seen that, particularly with the traumatic cerebral lesions of infancy, an immediate "recovery" without operative intervention may take place, and yet residual disturbances of serious import, appreciated only after some months, may remain. The same is true of adult lesions. Many of them doubtless are beyond the reach of our present methods of operative treatment, but as many more are within reach. The let-well-enough-alone policy is a wise one to follow under many conditions, particularly when it is evident that recovery from the immediate effects of an injury will take place without surgical interference; but when there is a likelihood that mental deterioration may follow such a "recovery," it is shortsighted to neglect any measures which may possibly lessen the probability of its occurrence.

Preventive measures are limited to the period immediately following the injury. Needless to say, when there is a palpable fracture of

the convexity it must receive attention, particularly when it is comminuted and when the dura has been injured. If there is doubt as to the presence of a depression it is better to explore and determine the fact. Subdural clots in particular must be carefully removed, and if possible a primary or a secondary closure of the dura should be made over a cortical laceration. Unless it will be in an obtrusive situation or on the forehead, it is better to err on the side of leaving a defect in the skull than otherwise.

More important are the cases without evident fracture, which are usually left to run their own course. This is particularly true of basal fractures: For when they are accompanied by subdural hemorrhages, or when acute edema is present, leading to compression symptoms, a decompressive operation with evacuation of fluid, bloody or otherwise, and the establishment of a permanent defect under one or both temporal muscles, often serves not only to promptly check the immediate symptoms, but distinctly lessens the liability of the late "neuroses." I am fully convinced that properly conducted operations are thus of the greatest prophylactic value. Both Bullard and Spiller, in their studies of the late consequences of cerebral injuries, have pointed out that the post-traumatic neuroses—other factors being equal—are less likely to follow in cases which have been subjected to operation than in those which have not.

Aside from operation in the acute stage, too great emphasis can not be laid on absolute rest and enforced quiet for a long period after the injury. These patients, like others, are apt to be hurried out of the hospital; and an attempt at too early a date to resume their former activities often results in a disastrous nervous breakdown.

Operations for residual symptoms in their late stages are often most disappointing in their results. There is often some subjective improvement after the removal of a scar, the repair of a defect, trephining under a local point of tenderness, etc.; but usually the same symptoms or others reappear and the patient again seeks a willing surgeon until operation becomes a habit with him. Though in isolated cases remarkable and sometimes unaccountable cures follow surgical measures, even in patients having pronounced symptoms of long standing, they are most exceptional.

4. **Cranial defects and their closure.**—Are cranial defects of themselves prejudicial to the subsequent welfare of the brain? This question is such a many-sided one that a definite answer for all cases can not possibly be given. There is a tendency on the part of many surgeons to regard the pulsating gap in the cranium as in a measure the cause of some of the distressing post-traumatic symptoms which have been recorded in the preceding sections, and hence to regard it as something to be solidly closed if possible.

Arguments may be advanced for and against this view. There is no doubt but that we see a great number of patients suffering from headaches, local discomforts, mental derangement, epilepsy, and the like, in whom, from loss of bone at the time of injury or from subsequent operations, there has resulted a defect too large for closure by natural processes of repair. Unless it can be shown that defects in general, including those not of traumatic origin, are attended with ill effects, and further, when they are of traumatic origin, that their closure leads to an improvement in existing symptoms, it is natural to infer that the symptoms accompanying a defect are merely an expression of the cerebral lesion which was coincident with the cranial injury.

One or more defects normally are present in a child's skull. We, with malice aforethought, purposely make defects in certain operations—as upon the Gasserian ganglion or when bone is removed for purposes of decompression. Kocher, indeed, goes so far as to believe that they exert a beneficial influence in cases of traumatic epilepsy, so that in certain instances he even advocates their establishment as a therapeutic measure. Further, defects even of large size and resulting from serious traumatic injuries may not be accompanied by any untoward symptoms.

I am myself inclined to the view that closure of a defect should be limited to those cases in which it is in an obtrusive situation and makes an unsightly deformity; to those in which local pain or tenderness promises to be lessened; or occasionally when the patient has an obsession in regard to its presence.

So far as the condition may affect, in one way or another, the progress of symptoms which result from the cerebral lesion, there may be advantages in having an opening. It is presumably a wound of the dura associated with the defect which is of chief consequence, for without this the loss of bone should have no more effect on the brain than has the fontanel of the infant's skull. When the dura has been injured as well as the brain, an attached scar will form, whether or not there be a solid bony covering; and it is debatable whether the irritation of such a scar is less when there is a defect which allows it to pulsate with the brain or when there is a solid covering to which it adheres. The restoration of a smooth and unattached dural surface is of chief moment, and if this can be accomplished it makes little difference whether the bone defect be closed or not. A neuroglial proliferation which may have its origin in a cortical scar will progress, whether there is an intact or open cranium. There are many, however, who believe that closure of a gap in the skull resulting from traumatism is of the highest importance. Stieda, voicing von Bramann, has recently reported a series of 48 cases from the latter's clinic in favor of this view.

Should a closure of the defect be considered advisable there are several measures open. At the time of the injury suspicious fragments may be left in place (*primary implantation*), with drainage as a safeguard should necrosis occur; oftentimes even a large, entirely dislocated fragment may partially or totally heal in place. If, owing to laceration of the scalp, the condition of the wound is such that replacement seems unwise at the time, we may wait until it has become covered with healthy granulations, on which the preserved fragment, after sterilization, may be replaced (*intermediary implantation*). Finally, if a wound which was compound has been allowed to completely cicatrize over the defect and it becomes desirable to close it, we may select one of two methods. By the *heteroplastic method* a shell of bone, with its overlying periosteum, is taken from elsewhere, as from the inner surface of the tibia, and implanted in the reopened wound. It is no longer a common practice to use heterogeneous materials for this purpose, as celluloid or silver plates, though nature sometimes endures their insertion with charitable tolerance. By the *autoplastic method* a flap is made, including the scalp and the underlying periosteum, together with adherent fragments of the outer table, which have been chipped away with a chisel or fine saw (Nicoladoni) as the flap is elevated. This flap is then rotated on its pedicle, so as to close in the freshly denuded bone defect. The surface from which the flap has been taken is left to heal slowly or it may be covered at any time by a skin graft. This is the so-called autoplastic method of Müller and König—an improvement on a method of closing defects introduced in 1884 by Durante, which made use of the principle of *autoplastic par glissement* introduced by Ollier.

THE TECHNIC OF INTRACRANIAL OPERATIONS.

The osteoplastic craniotomy.—Taking this operation as typical of the more difficult modern cranial procedures, the general principles of technic of cerebral surgery in general may be made to center about its description. Many of the views to be expressed are purely personal ones and are given with the full knowledge that the instruments and operative details found satisfactory to one individual may be entirely unsuited to the operative requirements of others. Special training and familiarity with a given set of instruments engenders facility; unfamiliarity, even with a better tool, awkwardness. Aware that I may be using to-morrow a different method from that to be described and which Da Costa has called a “combined method,” I nevertheless am assured that it is characterized by safety; and avoidance of operative accidents in the approach to a cerebral lesion is, after all, the matter of chief moment.

General methods of preparation.—There is no ward preparation beyond abstinence from food and insuring what should under other circumstances be a daily occurrence—a normal evacuation of the bowels. Patients are too often weakened and made uncomfortable by a purge which acts the morning of operation. The scalp, which may have been shampooed, is shaved just before the operation—a duty incumbent on the surgeon in case of a nervous patient or a child. There has never been an infection, even of a stitch in the scalp, in something over 300 cranial operations in the writer's series. (Many operators prefer to have the scalp shaved and treated antiseptically in the ward on the preceding day; some even advocate a double preparation.) After shaving there may be a preliminary cleansing of the scalp with soap and water and a soft brush, after which the head is wrapped in a wet bichlorid towel; the final preparation is deferred until after the anesthetic.

Position on the table.—It is a great advantage, though not a practice common to many, to place the patient on the table in the position most favorable for the operation before administering the anesthetic. It shortens the time as well as the depth of anesthetization, for a change of position from stretcher to table requires a degree of narcosis greater than needed for operative purposes; it insures a comfortable position for the patient and thus obviates the strains, backache, and so forth, which the handling of a relaxed body engenders. It is particularly important to observe this rule in case the cerebellum or occipital lobes are to be exposed.

Many operators have a particular form of headrest for all cranial operations, and Horsley, Frazier, Morestin, and others have described table extensions for this purpose. For the usual operations on the vault, however, I find that small, flat, solid pillows or sandbags are all that are necessary to turn and hold the head in the desired position; on the other hand, I find a head extension necessary for cerebellar work, chiefly to insure free respiration. Thus do surgeons' views differ. Whatever form of table be used, however, it is desirable to have the head end capable of being raised or lowered at will.

The anesthetic.—Regardless of the drug to be used, it is essential that it be administered by an expert, preferably by one who makes this his specialty. Many of the conditions for which these operations are done are associated with cardiovascular and respiratory disturbances of cerebral origin, and the greatest care must be exercised lest a further burden be imposed by the anesthetic.

In all serious or questionable cases the patient's pulse and blood pressure, first recorded in the ward, should be followed throughout the entire procedure by a *blood-pressure apparatus* and the observations recorded on a plotted chart. Only in this way can we gain any idea of physiological disturbances—whether given manipula-

tions are leading to shock, whether there is a fall of blood pressure from loss of blood, whether the slowed pulse is due to compression, and so on. A further necessity is an *artificial respiration apparatus*, to be immediately put into use in case there is failure of an already burdened respiratory center, either from the anesthetic, from loss of blood, or from additional compression due to cerebral manipulations.

Sir Victor Horsely, as is well known, is a strong advocate of chloroform combined with a preliminary hypodermic of morphin. Only a small amount of the drug is required, except during the early and closing periods of the anesthetization, and there is less likelihood of subsequent vomiting than when ether is used. It is taken smoothly, without cyanosis, and, furthermore, tends to lower the blood pressure; hence there is said to be less bleeding during the operation. In this country, where chloroform is doubtless administered less well than ether, the latter is the anesthetic of choice at most hands, the primary stage often being induced with ethyl chlorid. I believe, with Kocher, that there is an element of risk in the lowering of blood pressure by chloroform; and it is perhaps debatable whether this is not a more certain danger than the more active bleeding said to occur under ether; a drug which tends to hold the blood pressure high. Having had a fatality from chloroform, I use it far less frequently in cranial operations than formerly, restricting its use largely to children.

The question of the anesthetic in a two-stage operation is an especially serious one. Chloroform here would perhaps be less dangerous than a repeated etherization. Local anesthesia may at times be employed, though infiltration of the scalp is difficult. I have learned that no anesthetic whatsoever need be required for a second-stage operation limited to manipulations of dura and brain after re-reflecting the original bone flap.

Preparation of the operative field.—With the patient anesthetized and in proper position on the table, the final cleansing is done; for this alcohol and 1:1000 bichlorid solution suffice.

It is my practice at this stage, before the landmarks are obscured by the covering of operative sheets and towels, to outline the proposed flap on the scalp with a superficial cut of the scalpel. Those who have by long practice familiarized themselves with craniocerebral topography can mark out the main fissures on the scalp with no greater margin of error than when measurements are employed. Furthermore, as a large opening is to be made, the accurate determination on the scalp of the point overlying a given center is to-day less essential than formerly, when an attempt was made to approach it directly through a small trephine opening. Those who are less familiar with the topography of the brain will need to mark out these fissures by the aid of some one of the craniometers or rules

of measurement which have been described. As this is somewhat time consuming, many prefer with an indelible pencil to delineate these landmarks on the shaved scalp the day before.

With the proposed flap thus outlined and the head raised by a hand under the back of the neck, a broad square of wet bichlorid gauze is thrown over the entire head and over this is placed a *tourniquet*.

For the *control of hemorrhage from the scalp* numerous forms of tourniquet have been advocated. Many use a simple rubber tube or Esmarch bandage, both of which are difficult to apply and to fasten without slips in the aseptic technic; Crile uses a rubber dam over the entire scalp, which is thus rendered bloodless; I formerly used a pneumatic tourniquet, but have finally come to a form of rubber ring, which is snapped over the head from glabella to suboccipital region and which has a median tape to prevent its slipping over the eyes. This ring, with its tape of proper length, should have been measured before the operation, and, having been boiled, it is applied by the operator and an assistant. Usually all bleeding from the scalp is controlled by the tourniquet, though in certain cases of tumor with marked intracranial stasis some of the veins on the concave side of the incision, receiving blood from the skull, may have to be clamped. Special forceps have been designed for this purpose by Nicholson, Howzel, and Chipault. Other methods of controlling the vessels of the scalp have been advocated; as (1) preliminary ligation or temporary closure (Crile) of the carotid; (2) the blocking by three mass ligatures of tissue, including temporal, occipital, and supra-orbital arteries; and (3) the enclosure of the proposed incision by a running suture passed through the scalp on each side of it with the purpose of leaving an anemic band of tissue (Heidenhain).

With a proper arrangement of towels and, finally, a large operative sheet which covers the etherizer like a tent, the field is prepared leaving exposed little more than the area encompassed by the preliminary incision.

The osteoplastic flap.—In the line previously scratched on the scalp the incision is made through gauze and soft parts down to the skull, and when the bone is exposed it can be opened in a number of different ways. It may be recalled that in the original operation as described by Wagner (1889) the flap was outlined with mallet and chisel, and Chipault, Keen, and many others followed his lead. Keen long employed a particular form of angular gouge, with which the incision was quickly and skillfully made through the greater thickness of the skull, the flap being finally loosened by a few blows, which served to break such portions of the inner table as had escaped

division. Küster has recently advocated a chisel of different form for the same purpose.

Most operators object to these methods on the score of possible concussion even from the glancing blows which are given. Toison (1891) suggested the division of the bone with a chain saw passed between primary trephine openings, with cutting of the bone from within outward. This remains a first principle. Obalinski (1897) recommended for this purpose the flexible wire saw (introduced by Gigli, in 1897, for obstetrical purposes). A linear cut of 2 or 3 mm. width through the full thickness of the bone may be made with biting forceps of Montenovesi or Doyen pattern, or with the cutting hooks introduced by Dahlgren and De Vilbiss. These instruments progress slowly, but they are almost certain not to injure the dura and have the advantage of biting outward, so that there is little, if any, jar to the brain. Flexible wire saws may be used, that perfected by Gigli being the best. As it cuts from within outward and when taut straightens on the arc of the cranial circle, the saw must be guarded in order to protect the dura from injury. Similarly, the bone may be cut between the primary openings by sawing with a straight handsaw from without inward and Doyen has devised a special saw with a guard for this purpose. It is a dangerous tool in inexperienced hands.

There are, further, certain craniotomes which cut in a circular fashion by swinging the blade about a fixed point and which may be used to incise scalp as well as bone. The Stellwagen instrument (*Annals of Surgery*, 1902) has been highly praised by Philadelphia surgeons. A French instrument of similar type was described by Codivilla in 1900 (*Revue de chirurgie*). Though no preliminary trephine opening is needed, these methods possess the disadvantage of cutting from without inward, and as there is no guard, the dura is likely to be injured by these *craniotomes à mouvement circulaire*, unless the membrane is approached with the greatest caution.

The electromotor has been employed to furnish the driving power for a number of instruments which possess a circular or spherical form and so can revolve, such as burrs, trephines, and circular saws (of which there are a number of patterns, Van Arsdale's, Powell's, Marsland's, and Doyen's). It has led, furthermore, to the invention of a revolving tool or fraise with spiral cutting edges, which is used by some operators. Cryer, in 1897, Sudeck, in 1900, and Sykes have all described similar instruments of this type, and though useful for certain purposes they possess the disadvantage for osteoplastic work of cutting such a wide slot that the replaced flap subsequently rests on the dura instead of on the bone edges. In the Doyen type of motor there is a long flexible arm between the motor and the revolving tool, which is thus driven from a distance, as in the usual form of

dental engine. Borchardt (1906) has made some modifications of the apparatus, and Bercut (1904) and Hartley (1907) have made the further great improvement of having the cutting tool directly connected with the motor, which, weighing only 8 or 9 pounds and being capable of sterilization, is itself held in the operator's hands. Hartley has devised also new forms of perforators with serrated edges.

Surgeons who use electromotive force for osteoplastic operations are able to work very rapidly, and if this does not mean added risk of accident it is desirable. However, having witnessed, twice from a Doyen circular saw, and once from a Cryer drill, what I regard as a most serious accident—namely, the division of bone and dura at the same time, owing to the fact that the guide of the speeding instrument worked its way through the adherent membrane instead of separating it from the skull—I have clung to the somewhat slower, but certainly less dangerous operation by hand-driven instruments.

The methods of procedure which suit my own personal needs may be described.

The osteoplastic procedure.—This combines the following principles: Of *division between primary openings* (Toison); of incision by an *advancing instrument from a single opening*; of making *all cuts from within outward*; of leaving a *beveled flap*.

A primary opening through the thickest part of the exposed cranium, usually near the parietal eminence, is made with a hand trephine, which should be of generous size, with a crown of fully $2\frac{1}{2}$ cm. I prefer a trephine of the Galt conical pattern with a beveled edge; it is the safest instrument and obviates the necessity of an extractor (*tire-fond*) for the button, since, owing to the width of the superficial cut, it can easily be tilted out. In France the old form of cylindrical trepan is still in general use. Bleeding from diploëtic vessels in certain cases of tumor may be severe. A fatal case, indeed, of this sort has been reported by Ransohoff. It may be controlled by the proper use of Horsley's wax, with which the beveled teeth of the trephine may be filled. One or more secondary openings (one is usually sufficient) at the upper edge of the incision are made with a Doyen burr. With a long-handled blunt dissector or dural separator introduced through the large trephine opening the dura is separated from the bone between these openings. On withdrawing the dissector the intradural pressure suffices to press dura against bone again and thus to stop the bleeding. From the two trephine openings the lateral edges of the flap are then cut downward toward the base, in line with the skin incision. The first $\frac{1}{2}$ inch of these lateral cuts is made with Montenovesi forceps with a 3 mm. incision, followed by the Dahlgren forceps, as the thinner bone near the temporal region is approached. A Gigli wire saw is then passed on a guide (of which

there are numerous forms) between the two primary openings, and the mesial edge of the flap is cut on a broad bevel. (This is an important detail, for it enables the subsequent solid replacement of the flap without danger of its being driven inward by a snug pressure bandage.)

The flap is then forced back by the insertion of blunt instruments around the edges and is broken across at its base. As Hartley has emphasized, all flaps made on the cranial vault should radiate toward the temporal bone as a base, since this is the thinnest part of the calvarium and most easily broken. Provided the flap includes the region of the pterion, the meningeal artery may be torn, owing to its having channeled the broken bone. The vessel should be ligated at its lowest point of exposure by making at a distance a small opening in the dura, through which a grooved dissector is inserted and on which the curved needle should be passed to avoid the chance injury of some cortical vessel. Bleeding from the expansions of the lateral sinus, in case they have been exposed by a high flap, is best controlled by the pressure of sterile absorbent cotton, pledgets of which I find to be as valuable as a hemostatic agent for the intracranial part of the work as is wax for the bone itself. Horsley uses hot saline or weak bichlorid irrigations.

The intracranial procedures.—At this stage, if there has been a fall of blood pressure from loss of blood, the further progress of the operation, especially in tumor cases, may well be postponed for a second session. The question of a two-stage operation and the possibility of a second stage without anesthesia has been considered under the treatment of tumors. If there is no contraindication on this score, the dura is opened in a line concentric with the bone incision, leaving plenty of margin for subsequent suture. The membrane should be incised on a grooved director, especially if there is increased tension, lest the pia-arachnoid be injured. The incision should not be made too near the median line, lest the edges of the parasinoidal expansions, or the veins entering them, be wounded. If the mesial edge of the hemisphere is to be exposed, it is well to open the dura in this direction by a separate radial cut, and if necessary to rongeur away some of the bone toward the median line. This is preferable to an attempt at exposure of the mesial edge (foot area), through the primary osteoplastic flap—a procedure necessarily attended with a great loss of blood.

If the expected lesion is not disclosed and if the topography is not perfectly clear, the fissura centralis may have to be determined by faradization of the cortex. A long glass unipolar electrode carrying a fine platinum wire core, coiled into a spiral at the end according to Sherrington's plan, in order to obviate puncturing the pia-arachnoid, is used for stimulation. The other pole is attached to an ex-

tremity, preferably on the homolateral side. The current should be just strong enough to contract exposed muscle—some of the temporal fibers are usually available for this test. If there is an abundance of cerebrospinal fluid in the arachnoid spaces, it must be evacuated by pricking the membrane as it bridges the sulci; and, further, the patient must not be too deeply under the anesthetic. No motor cortex, unless there is complete degeneration of the pyramidal tract, fails to give responses if these precautions are observed.

If an incision of the cortex is necessary, whether for exploration, for extirpation of an area in focal epilepsy, or for the removal of brain tumors, the cortical vessels to be divided must first be ligated on each side of the proposed incision, which should, if possible, be confined to the exposed surface of a convolution and should not cross a sulcus. The finest strands of split silk, preferably black, should be used for these ligatures, and they should be passed around the vessels with delicate curved French needles, which are introduced and emerge in nonvascular areas. With these precautions the sub-cortical manipulations can usually be conducted with but little loss of blood, even in most cases of tumor extirpation. Tumors which have approached and involved the cortex must be surrounded by a similarly placed double row of ligatures, between which the incision is made. Subsequent dissections are made with blunt instruments, and momentary pressure of cotton pledgets will usually check the oozing.

A brain which tends to protrude may sometimes be dropped back by elevation of the head and trunk or by evacuating cerebrospinal fluid. This can at times be accomplished by opening the arachnoid spaces and by milking out the fluid; at other times a lumbar puncture may be necessary, and the removal of fluid in this way during the course of an operation is of great help under many circumstances, and is free from the danger which attends a similar proceeding when the skull is closed.

Closure.—Unless an irremovable growth indicates the necessity of permanent decompression, an accurate approximation of the dura in its two layers is desirable; this should be painstakingly done, to prevent the formation of adhesions of their re-formation if they have been found and divided, and have been productive of symptoms, as in epilepsy. If there is a large cortical defect, as after the removal of a growth, or if the brain has receded from its normal level, the space may well be filled with warm salt solution before closing the dura. The bone flap is solidly replaced and the scalp is in turn accurately approximated in a broad surface. It is well to draw together the galea aponeurotica by a few buried sutures before closing the outer layer. For the latter, many use a continuous suture, which

has the advantage of speed. Inasmuch as the closure in many cases is completed before the tourniquet is removed, I feel the need of a more accurate and solid approximation. This is done by rapidly placing about the incision a series of straight, round-pointed cambric needles, which serve to keep the edges everted as each suture is tied, and thus to assure a ridge of tissue with a flat approximation which prevents subsequent bleeding from the vessels of the scalp.

Drainage is occasionally advisable—perhaps in 20 per cent of the cases—but it should be avoided if possible. It is necessitated by constant oozing from the exposed parasinoidal sinuses, for otherwise an extradural clot may form. Marion drains directly through the center of the flap, in which a new opening has been drilled. I prefer to take advantage of the trephine openings already made at the upper angles of the flap. The drains, of cigarette form, covered with protective gauze so that they may be easily withdrawn, are led out, not through the original incision, but through puncture wounds made through the scalp 2 cm. to the outer side; this insures an oblique passage which can be occluded by pressure in case there should be a tendency for cerebrospinal fluid to escape after the drains have been withdrawn. Kocher has devised small silver tubes for drainage in similar fashion, and they serve the purpose admirably.

The wound is partly dressed and pressure is applied before the tourniquet is removed; then an abundant dressing with an outer starched bandage is employed. The ears should be carefully protected with cotton to prevent discomfort from pressure.

The first dressing is made in 48 hours, when the drains, if used, and all the sutures are removed.

Special operations.—Although these osteoplastic resections typify the methods of approaching the brain in a large percentage of our cases, they are not particularly applicable to operations elsewhere than upon the vault, nor are they necessary in cases in which from the first it is evident that bone must be permanently removed for decompressive purposes. Certain principles relating to these special operations may deserve mention.

Suboccipital operations.—For the exposure of subtentorial lesions (such as tumors of the cerebellum or of the lateral recess, a basilar meningitis to be drained, the freeing of adhesions about the fourth ventricle resultant to an old inflammation, etc.) the principles of tourniquet and bone flap are not applicable. In this situation, just as under the temporal muscle, owing to the possibility of subsequent firm closure under muscle, there is less reason for preservation of bone. Though many surgeons place the patient on the side for these operations, I much prefer a symmetrical, face-down position, particularly as a bilateral exposure is usually called for. This position interferes greatly with respiration unless the shoulders are held away

from the table so as to allow of free thoracic movements, and consequently, for these cases I have devised a table extension with shoulder supports and a separate crutch with a horseshoe-shaped top in which the forehead and cheek bones comfortably rest. The anesthetic is sprayed against a mask attached under the "horseshoe."

It has been mentioned in the section on tumors that a bilateral exposure of both cerebellar lobes is desirable to allow of dislocation outward of the normal lobe during the manipulations of the other. Hence I prefer a symmetrical form of approach, and find that a median incision, which divides the soft parts down not only to occiput, but to the spinous processes of the upper cervical vertebræ, in addition to the usual curvilinear cut over the occipital ridge ("cross-bow" incision, gives additional room, owing to the lateral reflection of the flaps. A fringe of muscle and aponeurosis together with galea is carefully preserved at the upper edge for subsequent union by suture with the reflected muscle flaps. The bone is bared and, after making bilateral primary openings, is rongeured away—upward, so as to expose the lateral sinus on each side; across the median line, leaving intact the bone over the torcular; and then downward, so as to include the posterior half of the foramen magnum. The dura is then widely opened and the midoccipital sinus, if present, is ligated.

This opening, through the possibility of cerebellar dislocation, gives a wide area of exposure, either of the fourth ventricle or of the structures in the cerebellopontine angle; and it is desirable, not only for exploration, but for decompression of incurable tumors in this situation, that it be bilateral. There are certain points in the bone where hemorrhage may be met with in these operations, and especial care must be taken at the torcular and at the approach to the mastoid processes. Intracranially a large vein often bridges across the subdural space at the side of the cerebellum, injury to which should be avoided if possible.

Temporal operations.—Operations either for decompression purposes, or in exploration for hemorrhage, tumor, or abscess in the temporal lobe, or for lesions in the middle cranial fossa, are conveniently carried out by simple splitting of the temporal muscle without division of the fibers. Here again, as in the occipital operation, there is no reason for the preservation of the bone, as there remains a secure muscular protection, which prevents too great bulging in case of increased tension, or an obtrusive and deforming depression in its absence. The incision through the scalp may be made parallel to the muscle fibers or as a curved incision across them. In removing the bone, flat-bladed rongeur forceps are necessary, as the muscle can not be lifted far away from the skull, and care must be taken not to injure the meningeal in case it lies in a canal at the lower angle of

the parietal bone. A large defect giving free exposure of the temporal lobe may be made in this way.

Should it be necessary to bring into view the base of the skull, as in operations upon the Gasserian ganglion or for tumors in its neighborhood, it is better to divide the muscle fibers without attempting a splitting operation.

These basilar operations through the temporal region may be carried so far inward as to expose the hypophysis. A view at this depth, however, requires such a degree of elevation of the brain that it is necessary to have a large cranial opening in order to allow of dislocation outward, which obviates, in a measure, the danger of compression effects. As conducted by Horsley in his remarkable series of cases, this is an intradural operation conducted, after making a wide opening in the skull, directly under the temporal lobe; after its exposure the new growth situated in the sella turcica is removed with the proper form of curet.

Frontal operations may be required for the exposure of lesions of the orbital or mesial surfaces of the frontal lobe or anterior end of the corpus callosum. An approach to the pituitary fossa has also been attempted by Krause directly under the frontal lobes after turning down a large frontal bone flap. Similar methods have been suggested by Kiliani (1904) and by Duret (1905), while Hartley (1907) advocates a bilateral flap with a pedicle in each temporal fossa. Here, too, a great deal of room for dislocation is necessary, and it would seem that the chance of frontal-lobe injury, far more serious than an equal degree of bruising of the temporal lobe, would make Horsley's route preferable.

These, like many other of the more serious and dangerous of the modern intracranial operations, should, for the time being and until their veriest detail is established on an unquestioned basis, rest in the hands alone of those specially trained in the conduct of cerebral operations. The advance of neurological surgery is greatly impeded by the prevailing impression in regard to its dangers and general futility—an impression due in large measure to the unsuccessful attempts of the untrained and inexperienced.

PART 3

THE VESTIBULAR APPARATUS IN RELATION TO THE DIAGNOSIS OF INTRACRANIAL LESIONS.

(From Equilibrium and Vertigo. By ISAAC H. JONES, M. D. Published by J. B. Lippincott Company.)

THE EAR AND THE NEUROLOGIST.

The intimate relation of the ear to the central nervous system makes this study of Neuro-otology of especial interest to the neurologist. The value of an eye examination in neurologic cases is now universally recognized. The study of the eye grounds, the field of vision, pupillary reaction to light and accommodation and the degree of function of the eye muscles, furnishes valuable information to the neurologist. Based on the opinion of the neurologists and ophthalmologists most acquainted with these ear tests, it would seem safe to assert that of the two methods of approach, very much more definite information can, as a rule, be had from the examination of the vestibular apparatus than from an eye examination. The value of ear examinations in neurologic cases is recognized in Vienna to the extent that no examination of a neurologic case is considered complete without a report from the otologist as to the condition of the vestibular apparatus.

The neurologist is not only concerned with the problems of equilibration which have already been presented, but, in addition, he has, in these ear examinations, a very definite help in many perplexing diagnoses. The ear tests are of particular value in making a differential diagnosis between labyrinth and intracranial lesions and in furnishing additional data in intracranial localization.

It is well known that nystagmus and vertigo, with loss of equilibration, associated perhaps with nausea and vomiting, may be produced either by a disturbance of the internal ear or by an intracranial lesion. In many instances the symptoms of internal ear disturbance and of a cerebellar lesion are identical. It not rarely happens that a careful neurologic study indicates a lesion of the cerebellum, whereas the ear examination, by giving additional data to the neurologist, demonstrates conclusively that he is dealing with a lesion of the labyrinth.

A differential diagnosis between peripheral and central lesions by means of the ear tests depends on certain general principles. A pe-

ripheral lesion of the labyrinth or VIII nerve is suggested by the following:

1. An impairment of the function of both the cochlear and kinetic-static labyrinth. If, for example, the hearing tests show cochlear deafness and the tests of the semicircular canals show that their function is also impaired, it immediately becomes probable that we are dealing with an end-organ lesion.

2. The history or presence of tinnitus; the absence of tinnitus does not necessarily indicate that the end organ is not involved, but its presence is very suggestive of labyrinth involvement.

3. Proportionate impairment of the responses from the horizontal canal and of the responses from the vertical canals. If for example, the tests show that the horizontal canal retains only one-half of its normal function and that the vertical canals similarly retain only one-half of their normal function, a lesion of the end organ itself is suggested.

4. Proportionate impairment of both nystagmus and vertigo. If the horizontal canal produces one-third of the normal nystagmus and one-third of the normal vertigo, it is suggested that the lesion is in the horizontal canal itself or in the fibers from the canal within the VIII nerve; if in addition the vertical canals produce one-third of the normal nystagmus and one-third of the normal vertigo, an end-organ lesion is strongly suggested.

In a word it is the "proportionate impairment" of responses that speaks for a peripheral lesion.

A *central* lesion is suggested by the following:

1. A normal cochlea but impaired or nonresponsive semicircular canals.

2. Normal responses from the horizontal canal but absent responses from the vertical canals.

3. Normal responses from the vertical canals but impaired responses from the horizontal canal.

4. Normal vertigo but impaired nystagmus from the horizontal canal.

5. Normal nystagmus but impaired vertigo from the horizontal canal.

6. Normal vertigo but impaired nystagmus from the vertical canals.

7. Normal nystagmus but impaired vertigo from the vertical canals.

8. Normal vertigo and normal nystagmus from any semicircular canal, but impaired *past pointing* in any direction of any one extremity.

9. Normal vertigo and normal nystagmus from any semicircular canal but an impairment or absence of the normal *falling*.

10. Spontaneous vertical nystagmus is pathognomonic of a central lesion and is indicative of involvement of the brain stem caused either by infiltration or pressure. A lesion of the labyrinth may produce many forms of spontaneous nystagmus—horizontal, rotary, oblique or a mixed nystagmus of the various types; but an ear lesion can never produce a spontaneous vertical nystagmus, either upward or downward.

11. If there exists a spontaneous nystagmus to the right and non-responsive semicircular canals of the right ear, an intracranial lesion is suggested. The nonresponsive labyrinth, if the labyrinth itself alone were responsible, would produce a nystagmus to the left.

12. A spontaneous nystagmus of increasing intensity or of long duration is indicative of a central lesion. A spontaneous nystagmus due to a lesion of the labyrinth shows its greatest intensity at the onset of the disease, becomes less and less marked, and disappears after a few days.

13. If a stimulation of any semicircular canal produces a “perverted” or “inverse” nystagmus, it is pathognomonic of a central lesion and is indicative of a brain-stem lesion. Such phenomena as the following are frequently seen:

Douching the right ear with cold water with the head back, stimulating the right horizontal canal, should produce a pure horizontal nystagmus to the left. If on such stimulation there occurs a vertical nystagmus upward or downward, a rotary, oblique, or mixed nystagmus, it may be spoken of as “perverted.” If instead of a horizontal nystagmus to the left there is produced a pure horizontal nystagmus to the right, it may be termed an “inverse” nystagmus. Neither a perverted nor an inverse nystagmus can possibly be produced by a lesion of the labyrinth or VIII nerve; a peripheral lesion produces a poor nystagmus or no nystagmus at all, but an absolutely false response of necessity demonstrates a central lesion.

14. If ear stimulation produces a conjugate deviation of the eyes instead of a nystagmus, it is pathognomonic of a central lesion.

The above outline indicates how additional data may be furnished to the neurologist by the ear tests in determining whether he is dealing with a lesion of the internal ear or of the brain stem or cerebellum. In the broader field of intracranial localization examination of the ear and of vestibular apparatus is also of distinct value. The particular feature of the ear examination is that the aurist sends in a *stimulus* to the brain centers, and then notes the *responses* of different parts of the body to this stimulus. For example, by stimulation of the ear there results a nystagmus in a given direction, a pointing of the extremities to the right or to the left, as the case may be, and a falling to the right, to the left, forward, or backward, as the case may be. Now, if the ear and these central paths from the

ear are intact, all the normal responses will appear; if there is a failure of all or any of the responses, it is positive evidence of an interruption along that particular path or paths that fail to bring about these responses.

In order to utilize the knowledge obtained from these tests it is essential to have in mind the various pathways constituting the vestibular apparatus. It may be stated at this point in brief that the pathways from the horizontal semicircular canal are different after entering the brain stem from those from the vertical canals; furthermore, that each set of tracts divides into two separate pathways; one pathway, the vestibulo-ocular tract, is responsible for the eye movement, and the other pathway, the vestibulo-cerebello-cerebral tract, conveys the impulses from the ear to the cerebral cortex, producing vertigo. If the horizontal canal fails to produce both nystagmus and vertigo the lesion indicated is at a point before the division of the vertical canal fibers into their two separate pathways. Further, if the horizontal canal produces normal vertigo but no nystagmus the lesion indicated is of the vestibulo-ocular tract at a point beyond the point of division into the two paths. If the horizontal canal produces normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into the two pathways. Similarly, if the vertical canals produce normal vertigo but no nystagmus, the lesion indicated is in the vestibulo-ocular tract at a point beyond division into the two paths. If the vertical canals produce normal nystagmus but no vertigo, the lesion indicated is at a point along the vestibulo-cerebello-cerebral path beyond the point of division into the two pathways. The ear tests have proven themselves surprisingly helpful in locating lesions in the cerebello-pontile angle, medulla oblongata, pons, cerebellar peduncles, cerebellum, and various portions of the cerebrum, including the parietal lobe, the temporal lobe, and the occipital lobe.

The value of an eye examination in neurologic cases is now universally recognized. The study of the eye grounds, the field of vision, pupillary reaction to light and accommodation, and the degree of function of the eye muscles furnishes valuable information to the neurologist. Based on the opinion of the neurologists and ophthalmologists most acquainted with these ear tests it would seem safe to assert that of the two methods of approach very much more definite information can, as a rule, be had from the examination of the vestibular apparatus than from an eye examination. The value of ear examinations in neurologic cases is recognized in Vienna to the extent that no examination of a neurologic case is considered complete without a report from the otologist as to the condition of the vestibular apparatus.

In order to obtain reliable data from an ear examination it is essential that the technic of examination should be accurate and painstaking; since it is primarily an ear examination the otologist is peculiarly fitted to carry out such examination. Although one purpose of this book is to furnish a practical guide for the otologist in undertaking the examination of patients, it is also offered to the neurologist so that he, on his part, may become familiar with the ear aspects of the work, in order to realize the significance of the reactions as reported to him. The ear examination is obviously not for the purpose of making a neurologic diagnosis; it merely gives additional information by a series of refined experiments, to the other methods at the command of the neurologist. To be sure, there are many cases in which the neurologist, without the aid of the ear, eye, blood, or other examinations, finds no difficulty in arriving at a satisfactory diagnosis. In these cases, however, it is of course useful to have the additional evidence from the ear tests corroborating his neurologic findings. In addition it not infrequently happens in obscure cases or in cases in which the neurologic data are meager that the ear tests may be the only means of furnishing information upon which a diagnosis can be made. For example, an apparently strong, vigorous man, complaining only of headache, showed on examination that both internal ears were normal and also that both VIII nerves were normal, and yet the vertical semicircular canals of both ears, when stimulated, failed to produce any responses whatever. The horizontal canals produced normal nystagmus, but no vertigo. As the labyrinths and VIII nerves in this case were unquestionably normal the nonappearance of the normal responses to stimulation could be accounted for only by an interference with the fibers from the labyrinth within the brain stem. This particular phenomenon complex indicates pressure within the IV ventricle. This conclusion was recorded with considerable misgiving because a lesion within the IV Ventricle appeared ridiculous in view of the man's apparent health. That night the patient was rushed to the hospital unconscious. The next day he regained consciousness, but complained of agonizing headache. Examination by a number of internists and neurologists failed to give any clue of an organic lesion anywhere, and the diagnosis of hysteria was made. Autopsy three days later showed abscess in the IV ventricle. It is in such cases as this that the ear examination is of the utmost importance, as it gives data absolutely unattainable by the usual neurologic tests.

THE EAR AND THE SURGEON.

The diagnosis of the precise location of lesions within the cranium is probably the most difficult task with which the surgeon is confronted. Fully realizing all the difficulties in these cases, he utilizes

every modern aid in diagnosis—the laboratory, the X ray, or any other instruments of precision that may be available. For many years the ophthalmologist has been of great help to the surgeon in his intracranial cases, and no one would think of operating on such a case without first having the ophthalmologist's report as to his findings. The previous chapter has shown how the ear tests may furnish aid of value in the accurate localization of lesions which involve any of the vestibular pathways, as well as in differentiating between labyrinth and intracranial lesions. A point worthy of special emphasis is that, with the aid of these tests, he is frequently enabled to determine whether a lesion is operable or inoperable. Many lesions of the medulla oblongata, pons, or cerebellar peduncles, which are inoperable by the very nature of their location, will show very pronounced cerebellar symptoms, and not infrequently the cerebellum is explored, in such cases, in the hope of finding a tumor near the cortex, and removing it. For such a differentiation, in many cases, no method can equal the accuracy of the Bárány tests. If, after turning and douching, there appears a normal past pointing of both extremities in both directions, it may safely be assumed that the cerebellum itself, is intact. This cannot be regarded as absolutely final, but is much more definite than any other known method for determining the integrity of the cerebellum. It is perfectly conceivable that a case showing almost normal past pointing, may apparently reveal a lesion when a cerebellar decompression is done. We have seen two such cases. Here, however, it should be explained that in both these instances the lesion proved to be a cyst which grew from the brain-stem and extended backward between the cerebellar hemispheres, without, however, destroying the substance of the cerebellum itself. Such cases give definite neurologic evidence of a cerebellar lesion, and at the time of operation, such a lesion is apparently discovered. There may have been present asynergy, manifested by hypertrophy, adiodokinesis and tremor, yet these symptoms or phenomena were produced by involvement of the fibers *on their way* to the cerebellum, or by pressure upon the cerebellum itself. If, then, the ear tests demonstrate normal past pointing, it is strong evidence that the cerebellum itself is not involved.

Tumors in the cerebello-pontile angle either originate from one of the cranial nerves in the angle, usually the VIII nerve, or have invaded the angle secondarily from the cerebellum or brain stem. These tumors invading the angle from other adjacent structures also usually involve the VIII nerve. Obviously then a careful study of the various portions of the VIII nerve gives direct insight as to conditions in the cerebello-pontile angle. In addition, in our experience

the ear tests have usually demonstrated the two following phenomena in cases of cerebello-pontile angle growth:

1. An absence of all responses from the vertical canal of the ear *opposite* the side of the lesion. Given a tumor in the right cerebello-pontile angle, the usual findings are as follows: The right ear gives no responses—the cochlea shows absence of all function, and the horizontal and vertical canals fail to produce any nystagmus, vertigo, past pointing, or falling. The left ear shows unimpaired hearing and the left horizontal canal does produce nystagmus, vertigo, past pointing, and falling. The left vertical canals, however, fail to produce any responses, because the vertical canals fibers are most probably impaired because of pressure upon the pons by the tumor in the angle.

2. "Crossed past pointing." This phenomenon consists of persistent past pointing of both upper extremities either outward or inward, regardless of the type of ear stimulation employed.

If ear stimulation produces normal nystagmus, vertigo, past pointing, and falling, the Bárány tests are of unquestioned value in eliminating lesions of the posterior fossa and brain stem. If they rendered no other service than this it would be sufficient to hail them as a distinct contribution in the diagnosis of intracranial lesions. In addition these tests may prove helpful to the surgeon in preventing unnecessary operations. One case is of interest in this connection. A woman appeared to have a tumor of the right cerebellar hemisphere. This diagnosis was confirmed by neurologic consultants and the X-ray report stated that it was a cyst in the right cerebellar hemisphere. The ear tests suggested that the cerebellum was uninvolved; operation was delayed on this account and eventually was found to be unnecessary, as the patient steadily improved and was discharged from the hospital apparently well, and for years has had no recurrence of her symptoms. Her cerebellar symptoms evidently were due to a disturbance of the internal ear.

No operation upon the brain should be undertaken without giving the patient the benefit of an ear examination.

VESTIBULAR VERTIGO.

Vertigo, from whatever cause, is a subjective sensation of a disturbed relationship to space. This disturbance is necessarily due to an alteration, either stimulating or depressing, of some portion of the *vestibular apparatus*. Inasmuch as the tests of the static labyrinth disturb the vestibular apparatus, vertigo is necessarily a resulting phenomenon. It is distinctly a cerebral disturbance resulting from impulses carried from the ear to the cerebral cortex. The tracts producing vertigo run from the ear, through the VIII nerve,

through the brain stem, and through the cerebellar peduncles as far as the cerebellar nuclei. There are two separate afferent paths from the ear to the cerebral cortex, resulting in vertigo from ear stimulation: (1) The horizontal canal tracts and (2) the vertical canal tracts.

THE PLANES OF VESTIBULAR VERTIGO.

The vestibular reaction of vertigo, just like the reaction of nystagmus, manifests itself in certain definite planes:

- (1) Sensation of turning in the horizontal plane, either from the right to the left or from the left to the right.
- (2) Sensation of turning in the frontal plane, consisting of a sensation of *falling* to the right or falling to the left.
- (3) Sensation of turning in the sagittal plane, consisting of a sensation of *falling* forward or backward.

The sensation of movement in the horizontal plane is produced only by the horizontal canal or canals. This is illustrated by turning the patient with the head in the upright position.

The sensation of turning in the frontal plane is produced only when the vertical canals are influenced in the frontal plane. When the patient is turned with the head forward or backward, the plane of the head is frontal.

Now, if after turning the head is kept forward or backward, the subjective sensation is of turning in the frontal plane, which in this position of the head is *parallel to the floor*. The sensation is, therefore, the same as after turning with the head upright, namely, a movement about one's own axis either to the right or the left. As it is a sensation of turning in a plane parallel to the floor, it is not unpleasant. If, however, after turning, the patient's head is raised to the upright position, the frontal plane then becomes at *right angles* to the floor and the sensation is that of falling in the frontal plane either to the right or to the left, and is, therefore, incidentally unpleasant.

The sensation of turning in the sagittal plane is produced only when the vertical canals are influenced in the sagittal plane. When the patient is turned with the head inclined well over toward the shoulder, the plane of the head is sagittal. Now, if after turning the head is kept in this same position toward the shoulder, the subjective sensation is of turning in the sagittal plane, which in this position of the head is *parallel to the floor*. The sensation is, therefore, the same as after turning with the head upright, namely, a movement about one's own axis either to the right or to the left. As it is a sensation of turning in a plane parallel to the floor, it is not unpleasant. If, however, after turning the patient's head is raised to the upright position, the sagittal plane then becomes at

right angles to the floor and the sensation is that of falling in the sagittal plane; this consists of a feeling of pitching forward or backward, and is, therefore, incidentally unpleasant.

In speaking of this unpleasantness resulting from endolymph movement in the vertical plane, we may use the illustration of seasickness. Bárány calls attention to the fact that a ship at sea tosses in various *planes*:

(1) The horizontal plane, from right to left. This movement, however, is usually very slight and, unfortunately, as we have already shown, this is the only plane of movement that is not unpleasant.

(2) The frontal plane—namely, a rolling of the ship from side to side. If the individual is standing facing the bow of the ship, the vertical canals are affected in the frontal plane. This is unpleasant. If, therefore, the individual lies down with his head or his feet toward the bow, the rolling movement then affects his horizontal canals, and the unpleasantness disappears.

(3) The sagittal plane—namely, a pitching of the ship fore and aft. If the individual is standing facing the bow of the ship, the vertical canals are affected in the sagittal plane. This is unpleasant.

If, therefore, the person lies down with the line of the body extending across the ship from starboard to port, the pitching movement then affects the horizontal canals, and the unpleasantness disappears.

The up-and-down movement of the ship, rising and sinking, in a similar way affects the vertical canals when the individual is in the upright position. The unpleasantness caused by this movement is also relieved when the individual lies down, as then the up-and-down movement affects the horizontal canals, the stimulation of which is so much less unpleasant.

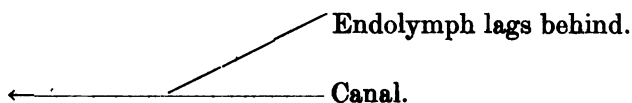
Unfortunately the movements of the ship at any given time are seldom in one plane only. If it were not for this, these suggestions of Bárány would unquestionably prove of great relief to the seasick. Even as it is we have the explanation of the fact that a seasick individual feels much better when he lies down.

THE ARTIFICIAL PRODUCTION OF VESTIBULAR VERTIGO.

The subjective sensation of vertigo after ear stimulation is due to the movement in one direction or the other of the hair cells of the end organ in the labyrinth; such sensations can be produced either by turning an individual in a revolving chair or by douching the ears.

Vertigo after turning.—If a person is turned with the head upright toward the right, with eyes closed, his first sensation is of turning toward the right. This is due to the lagging behind of the endo-

lymph in the horizontal semicircular canals. Diagrammatically this can be represented as follows:



Now, as the turning is continued the endolymph catches up to the movement of the body and the individual no longer *feels* that he is turning, although actually he is turning.



On stopping the chair the endolymph continues to move, and the person has a sensation of turning in the opposite direction, namely, to the left, although he is actually sitting absolutely quiet in the chair.



The essential feature of the subjective sensation of vertigo after turning is that after turning to the right the individual feels he is turning to the left, *regardless of the position of the head*. After turning to the right with the head forward, or after turning to the right with the head backward, although the movement of the endolymph is in a diametrically opposite direction, the result is the *same*—namely, a subjective sensation of turning to the left. It will be recalled that this is not true of nystagmus. Turning to the right with the head forward produces nystagmus to the *left*; turning to the right with the head backward produces nystagmus to the right. This is because nystagmus is a simple reflex, depending directly on the direction of the endolymph movement. Vertigo, on the other hand, is a cerebral phenomenon; in determining the interpretation of the sensation of movement, the cerebrum *takes into consideration the position of the head*, and in this way properly interprets the significance of the endolymph movement with the head in any given position.

The following is a table of the subjective sensations of vertigo produced by turning:

Turning to the right with the head upright produces a sensation of movement to the left in the horizontal plane.

Turning to the left with the head in the upright position produces a sensation of turning to the right in the horizontal plane.

Turning to the right with the head forward 120° produces—

(a) With the head kept in the forward position, a sensation of turning to the left in the horizontal plane.

(b) With the head brought upright, a sensation of falling to the left in the frontal plane.

Turning to the left with the head forward 120° produces—

(a) With the head kept in the forward position, a sensation of turning to the right in the horizontal plane.

(b) Bringing the head to the upright position, a sensation of falling to the right in the frontal plane.

Turning to the right with the head back 60° produces—

(a) With the head kept in this backward position, a sensation of turning to the left in the horizontal plane.

(b) With the head brought upright, a sensation of falling to the left in the frontal plane.

Turning to the left, head back 60° produces—

(a) With the head kept in this backward position, a sensation of turning to the right in the horizontal plane.

(b) Head brought to upright position, a sensation of falling to the right in the frontal plane.

It is not necessary to memorize all the subjective sensations of turning. In the chapter on nystagmus it is noted that the eyes are always drawn in the direction of the endolymph movement. As regards the subjective sensation of systematized vertigo, the following rules should be remembered: *Vertigo is always in the direction opposite to the endolymph movement.*

Experience by sight and muscle sense has taught the individual that when he turns to the right and the endolymph is lagging behind, that he is turning to the right. When he stops and the endolymph continues to go to the right, he therefore has a subjective sensation of turning to the left. The original basis for his mental interpretation of the endolymph is that when he turns to the right the endolymph moves relatively to the left. The mental interpretation, therefore, is always of a subjective sensation of movement in a direction opposite to the endolymph current.

Vertigo after douching.—On douching the ear with either cold or hot water, after a sufficient time has elapsed to permit of chilling of the outer wall of the labyrinth, there occurs a systematized vestibular vertigo. The following is a table of the subjective sensations of vertigo produced by the caloric test.

Douching the right ear, head upright, water 68° , produces sensation of falling to the left in the frontal plane.

Douching the left ear with the head upright, water 68°, produces sensation of falling to the right in the frontal plane.

Douching the right ear with the head 120° forward, water 68°, produces sensation of falling to the left in the frontal plane.

Douching the left ear with the head 120° forward, water 120°, produces sensation of falling to the right in the frontal plane.

Douching the right ear, head back 60°, water 68°, produces sensation of falling to the left in the frontal plane.

Douching the left ear, head back 60°, water 68°, produces sensation of falling to the right in the frontal plane.

Douching the right ear, head upright, water 112°, produces sensation of falling to the right in the frontal plane.

Douching the right ear, head back 60°, water 112°, produces sensation of falling to the right in the frontal plane.

Douching the left ear, head back 60°, water 112°, produces sensation of falling to the left in the frontal plane.

Douching the right ear, head forward 120°, water 112°, produces sensation of falling to the left in the frontal plane.

Douching the left ear, head forward 120°, water 112°, produces sensation of falling to the right in the frontal plane.

THE TECHNIC OF EXAMINATION OF THE STATIC LABYRINTH.

(By ISAAC H. JONES, M. D., and LEWIS FISHER, M. D. *Annals of Otology, Rhinology, and Laryngology*, March, 1917.

So little has been known of the physiology of the static labyrinth until comparatively recent times, that the lack of a well-established technic of its examination is not surprising. The value of the contributions of Ewald, Högjes, Von Stein, Alexander, Naumann, Shambaugh, E. R. Lewis, J. G. Wilson, Ruttin, and others is now, however, beginning to receive the recognition it deserves. By far the greatest impetus to this work was given by Robert Bárány, who in the last decade brought out the "caloric" and "pointing" tests—contributions which brought him the Nobel prize. With these tests came the realization of the intimate relation of the internal ear to the rest of the central nervous system. We know now that the static labyrinth is only the labyrinth of the vestibular apparatus—this vestibular apparatus consisting of the static portion of the internal ear and nerve paths connecting it with nerve centers in the brain stem, cerebellum, and cerebrum.

Originally tests of the labyrinth were carried out for the sole purpose of determining its own integrity. Such tests were sufficient for the aural surgeon who was called upon to decide whether or not to operate on the "end organ." With the development of the idea that the labyrinth is only one portion of the vestibular apparatus,

came the realization that this whole apparatus was being tested at the same time that the internal ear itself was being tested, and that an intelligent interpretation of the phenomena obtained by such tests can also give the examiner an insight into the condition of those various brain paths and brain centers in relation with the internal ear. Thus, when a known stimulus is applied to the labyrinth, any response obtained therefrom, be it nystagmus or vertigo, indicates not only a functioning and reacting labyrinth, but also intact pathways from the labyrinth to the brain centers responsible for those reactions. Conversely, the nonappearance of any of the normal responses to stimulation indicates an interruption at some point along the particular pathway that fails to produce that particular response. This made it evident that the old technic of examination of the static labyrinth was very inadequate, and Bárány elaborated a new technic. Recent investigations by writers in the department of neurotology at the University of Pennsylvania, under the service of Dr. B. Alexander Randall, have shown that the fibers from the so-called horizontal semicircular canal have an entirely separate course in the brain stem from those of the vertical semicircular canals, and while they all go to the cerebellum, we consider that those from the horizontal canal reach it by way of the inferior cerebellar peduncle, while those from the vertical canals go by way of the middle cerebellar peduncle. Bárány's technic, elaborated before such a differentiation was demonstrated, deals with the labyrinth as a whole, whereas in the light of this new knowledge, it at once becomes evident how important it is to examine each set of canals separately.

We also became convinced that nystagmus and vertigo are distinct and separate phenomena, and that, furthermore, the "past pointing" of Bárány is not a "cerebellar pull," but is a cerebral phenomenon exclusively and depends entirely on the vertigo induced by the ear stimulation. This again necessitated the modification of some of the existing tests as well as the employment of some new ones. Above all it is of prime importance that the technic be accurate, complete, and painstaking, if the data obtained from such an examination are to be relied on. We would suggest that those who appear to be doubtful of the value of these tests must ascribe their disappointment to laxity in methods of conducting them. This point has been impressed on us by the statement of one of the leading men in the profession, who claims that the "Bárány tests" had been routinely employed in his cases and proved of no value. We found that he had used no turning chair. Another authority reported a case in which there was an absence of the normal past pointing, and yet operation revealed a normal cerebellum. We found that this physician did have a turning chair, but that it was not equipped with a "stop pedal;" we have frequently seen cases examined without a

stop pedal in which no past pointing was obtained, and on testing them with our chair with the stop pedal, we were able to produce past pointing of a foot or more.

The technic about to be described has been employed in the examination of hundreds of cases, and the deductions drawn from them have been verified in many instances by operations and autopsies.

When we first began examinations of the static labyrinth, a great difficulty was encountered in the recording of the findings, or rather in the lack of recording them. All who have done this work realize what an unspeakable nuisance it is to keep haphazard records, on slips of paper, perhaps, of the results obtained from the various vestibular tests. For example: "On turning to the right the patient's nystagmus was horizontal to the left of so many seconds, but his past pointing was so much for the right arm to the right, and so much for the left arm to the right," etc. We all know how hopeless it is to attempt the analysis of a case from such records. To obviate this difficulty we gradually formulated the accompanying chart, in which all the tests are outlined in the order in which they are usually undertaken and which is so arranged that when properly filled in it shows all the vestibular data simply by a glance at one page. We think this chart is of so much help in the examination and diagnosis of a case that it is actually a part of the technic, and we will, therefore, describe it.

One side of the chart is devoted to miscellaneous details or such routine matters as are found on any chart, with particular emphasis on the examination of the cochlea. The other side is devoted exclusively to the vestibular tests, of which there are three divisions, as follows:

1. Spontaneous.
2. Turning.
3. Caloric.

There is a complete column for the "nystagmus," and another complete column for "pointing." This enables us to study the nystagmus as such by running the eye down the column to the left, showing first the spontaneous nystagmus, then the nystagmus after turning, and then after the caloric test. In the same way the pointing tests can be studied as such by following right down the page, first the spontaneous, then those after turning, and then after douching.

As a further aid there is a space reserved on the chart for summarizing the findings obtained after each form of examination. As we always look for the following phenomena—nystagmus, vertigo, past-pointing, and falling—they are indicated concisely, one underneath the other, in each one of the three subdivisions. The examiner, after he has turned the patient, simply summarizes by indicating

"nystagmus normal" or "absent"; vertigo, either "normal" or "subnormal," or "exaggerated" or "absent," as the case may be, and so on in the same way under the other headings. When the chart is filled in as we have indicated, and an analysis of the case attempted for the purpose of diagnosis, all the examiner need do is to look at these different summaries under the three main subdivisions to get a bird's-eye view, as it were, of the whole case.

We will imagine we are examining a patient and conducting the tests step by step, with the aid of the chart. The "miscellaneous side" of the chart is filled in first, taking up every heading indicated and filling it in with appropriate information—the name, the age, and so on, with emphasis on the history of dizziness, for the reason that there can be no impairment of the vestibular apparatus without dizziness as a symptom. The duration of the dizziness, its character, when it was first noticed, whether it came on gradually or suddenly, whether the attacks came on with sudden change of position—as, for example, on getting out of bed in the morning, or when washing the face—whether nausea or vomiting accompanied it—all are noted.

Staggering.—Did it come on suddenly or gradually? Is it constant or does it come on intermittently? Is its presence coincident with dizziness? Is it at any time severe enough to make the patient fall? If staggering and falling are present, is it always in the same direction?

Tinnitus.—The presence or absence of tinnitus is of considerable importance, since it might be an aid in the differential diagnosis between peripheral and central lesions. We would expect tinnitus to be present in affections of the labyrinth itself or in lesions of the so-called "end-organ" type, whereas it would be more likely to be absent in those disturbances of the vestibular apparatus located within the brain.

Deafness.—Its duration, and whether the loss of hearing was gradual or sudden, are noted.

Nose and throat.—A routine examination is made with a view of discovering some evidence of focal infection which might account for the presence of an irritative disturbance of the vestibular apparatus, should the rest of the examination point that way; also, for any abnormality which might throw some light on any possible intracranial condition, such as palsies of the tongue, pharynx, or vocal cords, anesthetics, or loss of the sense of taste or smell.

THE EAR.

We note the following:

1. Configuration of the auditory canals.
2. The presence or absence of a mechanical obstruction in the canals, congenital or acquired; of the latter there may be impacted cerumen or other débris or polypi.

3. The presence of inflammation or suppuration.

4. Condition of the tympanic membrane. The length of time necessary to douche before a reaction appears may depend in a measure on the thickness of the drumhead. Perforations are looked for, since a "dry perforation" would be a contraindication to douching.

5. Hearing function. This is of the utmost importance in all cases, because a knowledge of the condition of the cochlear labyrinth is frequently of the greatest help in the differentiation between an "intracranial" and an "end-organ" lesion. The cochlea is phylogenetically the younger and newer portion of the labyrinth, and, therefore, the weaker and less resistant to the action of toxins. A perfectly functioning cochlea would, therefore, per se, presuppose a normal static labyrinth.

The fistula test.—This is performed only in cases where there is a chronic suppuration of the ear and can be carried out with the use of the Politzer bag.

We are now ready for the vestibular tests, all of them being outlined on the "vestibular" side of the chart.

SPONTANEOUS PHENOMENA.

NYSTAGMUS.

The patient is instructed to look straight ahead of him at a distant point, as the effort at convergence when looking at a near object may obscure the findings to the extent of limiting or entirely effacing a nystagmus. The use of convex lenses serves three purposes: First, convergence is entirely impossible; second, the observer is able to view the eyes from in front; and third, the eyes are now much magnified. A spontaneous nystagmus of any form on looking straight ahead is always pathologic. The patient is then told to look to the extreme right and the extreme left. With an unintelligent patient this test is accomplished more easily by placing him in the revolving chair and have him fix his eyes on some distant point. The chair is then turned, first to the left and then to the right. In this way we revolve the body, as it were, around the eyes. A certain amount of lateral nystagmus, when looking to the extreme right or extreme left, is physiologic. It is pathologic only when it is of considerable amplitude or when there is a sharp difference between the nystagmus on looking to the right and on looking to the left. All forms of nystagmus become more pronounced when the individual attempts to look in the direction of the nystagmus. It is for this reason that the subject is made to look in various directions, as by so doing we are often able to bring out a pathologic nystagmus of an amplitude

not large enough to become evident on looking straight ahead. Nystagmus is recorded by means of an arrow—a straight one for horizontal nystagmus and a curved one for the rotary; it seems simpler, as suggested to us by George Mackenzie, to have the arrow point in the direction in which the examiner sees the nystagmus on the patient. With eyelids held wide apart, the patient is told to look directly upward and then downward, with a view of discovering a vertical nystagmus, if present. This is of the utmost importance for the reason that, as Bárány has pointed out, a spontaneous vertical nystagmus is invariably intracranial in origin; furthermore, in our experience a spontaneous vertical nystagmus has proven itself to be a pathognomonic symptom of involvement of the brain stem either by pressure or infiltration. If a vertical nystagmus is present it is recorded—the arrow pointing either up or down, as the case may be. Paresis or paralysis of any of the ocular muscles, as well as the ability to perform conjugate movements of both eyes, are noted.

SPONTANEOUS VERTIGO.

The patient is asked not merely whether he is “dizzy,” but whether he has any sensation of turning. If the latter is present, careful inquiry is made whether or not the turning is systematized. By systematized vertigo is meant:

1. The patient feels he is going in a certain direction.
2. The outside world is moving in a certain direction around him, but he himself is remaining still.
3. He himself is moving in one direction, and the world about him is moving in the opposite direction.

SPONTANEOUS POINTING.

The patient sits in the chair and is told to put his arm forward and point with his forefinger. The examiner holds out his own forefinger, and allows that of the patient to come in contact with his. The examiner steadies his arm against his own body and further steadies his outstretched forefinger by means of the other hand. The patient's forefinger is allowed to come in contact with the examiner's finger, after which the patient raises his arm, without bending the elbow, to the perpendicular position, and immediately brings it back to touch the examiner's finger. He is then directed to repeat this with his eyes closed. Should the patient find the finger, it is recorded under the column for pointing for the appropriate arm with a letter “T” (touched). Should he deviate either to the right or the left, the test is repeated a number of times to make sure that the failure is not the result of pure inattention, but that the deviation is constant and persistent in a certain direction. The distance of the

deviation is recorded in inches, either to the right or to the left, as the case may be, by noting under the appropriate pointing column—let us say “2 to R.,” if the deviation happens to be 2 inches to the right, or “2 to L.,” if the deviation happens to be to the left, etc. Should the patient fail to find the finger and “past point” either to the right or to the left, his arm must never be pulled over toward the examiner’s finger if the test is to be repeated, because if this is done the patient finds out that he “past pointed,” and might make a conscious correction to overcome an actual tendency to past point.” Instead, the examiner should again place his own finger under that of the patient. In taking the pointing, it is usually sufficient to test the pointing of the shoulder joint “from above,” as described; the other forms of pointing, such as “shoulder from below,” or “shoulder from the side,” or pointing of the hips, are not performed routinely—these are undertaken only in cases where such extensive examinations appear to be necessary.

SPONTANEOUS FALLING.

The Romberg test.—The patient is told to stand with heels and toes together and eyes closed. His station is noted. While in this position the head is quickly turned to the right, and observation made whether that had any effect upon the “station” or upon the direction of the patient’s “falling,” should he have any. The same observation is made after the head is quickly turned to the left. This test is of value in that patients with intracranial lesions always fall in the same direction, regardless of the position of the head, whereas a labyrinthine lesion causes falling in the direction of the affected ear.

“The attempt to overthrow.”—This is Bárány’s own “pelvic girdle test.” The individual stands as before, with eyes closed, heels and toes together. The examiner grasps the patient at both shoulders, and attempts to overthrow him either to the right or the left, forward or backward, instructing the patient all the while to resist this attempt at overthrowing by a balancing movement at the hips. When the shoulders of the patient are moved toward the right, the pelvis should sway toward the left in an attempt to maintain equilibrium. On pushing the patient backward, the pelvis should move forward, and so on. In this way the degree of freedom of pelvic movement is observed. A normal individual balances perfectly, and lean in any of the four directions considerably before falling, whereas in one with an affection of the vermis of the cerebellum, the pelvis fails to compensate, and the patient falls over like a broomstick at the slightest touch.

Goniometer.—This consists of a movable platform so arranged that it can be tilted out of the horizontal plane. One end of the platform

has a cord attached to it by means of which that end can be pulled down—the other end rising against a graduated perpendicular column. The patient stands in the center of this platform, either facing the examiner or at right angles to him. He is first tested with eyes open. One end of the platform is gradually lowered by pulling on the cord until the patient begins to sway, showing that he is about to lose his equilibrium. The other (perpendicular and graduated) end shows the number of degrees of tilting that were necessary before the patient began to lose his equilibrium. He is then tested in the same way with eyes closed. By means of the goninometer it is sometimes possible to bring out a latent tendency to fall not demonstrable in any other way. This is only occasionally useful.

Having completed the examinations for the various spontaneous phenomena, we are now ready to examine the vestibular apparatus by means of the turning and caloric tests. In order to carry out a good technic, it is well to bear in mind the whys and wherefores of the various tests. Both the turning and caloric tests depend upon the setting in motion of the lymph within the labyrinth. This circulation of the lymph stimulates the hair cells in the ampullæ of the semicircular canals, starting impulses which are transmitted by nerve paths to the corresponding centers in the grain. The principle underlying these tests is that a known stimulus applied to a normal labyrinth will produce definite phenomena if the nerve paths from such a normal labyrinth to their centers are all intact.

Before drawing conclusions, therefore, we must be certain of our technic, we must make sure that the proper stimulus was applied, and must forever bear in mind the particular nerve path we are employing in a given test. The following points should be remembered:

- (1) The desirability of testing only one set of canals at a time.
- (2) In the turning chair we test only those canals which are in the horizontal plane, or rather those out of the absolutely vertical plane.
- (3) The caloric test affects only those canals which are in the vertical plane, or rather those which are out of the horizontal plane.
- (4) Each canal, when stimulated, produces a nystagmus and a vertigo in its own plane.
- (5) The eyes are always drawn in the direction of the endolymph movement (this is the slow component).
- (6) The vertigo is always in the direction opposite to the endolymph movement.

The individual is placed in a smoothly revolving chair which has an adjustable headpiece, so that the head can be comfortably fixed and held in any desired position. The chair must also have a mechanism whereby it can be instantly stopped by means of a foot pedal

in a given position and rigidly held there. Without such an arrangement past-pointing tests can not be made accurately. Bárány constructed a special chair for this purpose, and we were fortunate enough to receive one of these chairs only a few days before the outbreak of the war in Europe. As it was hardly probable that any more of these chairs could come out of Vienna for some time, several physicians interested in the work asked us to design a chair along similar lines. After we had used the Bárány chair for several months the following changes suggested themselves and were incorporated in the new chair.

(1) The back of the chair and the headrest are so constructed that the patient's head is placed immediately over the axis of turning. This is obviously of great importance. In the original Bárány chair the head revolves away from the center of turning, and describes a circle with a diameter of over a foot.

(2) It is impossible in the Bárány chair to hold the head in a forward position. We therefore constructed an extra headpiece to permit rotation with the head inclined forward. It is obvious that it is absolutely essential for the head to be held steady when being rotated in this position, and this can not be accomplished without a special head bracket.

(3) Instead of having a special handle for turning, as in the Bárány chair, the rod at the back of the chair is made slightly longer and a handle placed at the top. The extra handle was very annoying and interfered with the pointing tests of the right arm by its presence on the side of the chair.

(4) The base is made much heavier than in the Bárány chair, in order that the patient may be rotated rapidly, if necessary, and yet not have the chair wobble; this gives both the patient and the doctor a greater sense of security.

(5) The Bárány chair is bound together by a great many bolts. In order to take the chair apart, in case it is desired to move it, all these bolts must be undone. In the new chair there are no bolts and the parts are all welded; the chair consists of only two pieces—the seat and the base. This makes it more portable, and is a great convenience when a patient has to be examined at a place where no such chair can be had. In constructing this chair we also aimed to make it suitable for use as a regular office treatment and operating chair, so that it would not require any extra room in the office.

TURNING.

The routine method is to test first for nystagmus, then for vertigo, and then for past-pointing. The horizontal canals are tested in the turning chair. To accomplish this the head must be secured in the

headrest, tilted 30 degrees forward—the reason being that with the head perfectly erect, the external or so-called horizontal semicircular canal slants 30 degrees backward. If this precaution is neglected, the vertical canals also enter into the reactions.

NYSTAGMUS AFTER TURNING.

During the turning the patient's eyes are closed. The chair is rotated to the right 10 times, at the speed of 2 seconds to each turn, making 20 seconds for the 10 turns, after which it is stopped. The patient is told to open his eyes and look off at a distance. The "after-turning" nystagmus is then noted, including its direction, character, and duration. The time of the nystagmus is best taken with a stop watch, the watch being clicked and time counted from the moment the chair is stopped until the very last nystagmus twitch disappears, when it is clicked again and the number of seconds read off. It is recorded with a horizontal arrow pointing in the proper direction after the words "to right" on the chart. The amplitude is recorded in terms of either "large," "small," "fair," or "barely a twitch." The duration is recorded in seconds as read off from the watch. In a normal case this nystagmus should be 26 seconds.

In a similar manner the patient is turned to the left.

The employment of the stop pedal is not necessary in testing for

VERTIGO AFTER TURNING.

Vertigo after turning may be tested quantitatively. The plane of the induced vertigo is always in the plane of the canal stimulated and in a direction opposite to the endolymph movement. If it is the horizontal canals we wish to test, the patient's head must be fixed with the chin tilted 30 degrees forward. He is then turned, with eyes closed, to the right at a uniform speed, and is asked to keep on telling the examiner in what direction he is being turned. Thus he keeps on saying "to the right, to the right." After 10 turns in 10 seconds the chair is stopped and immediately he will say, "I am going to the left." The stop watch is started the same instant and kept running as long as the patient thinks he is going to the left. When he says, "I am standing still," the watch is stopped and the reading of the duration of the vertigo taken in seconds. In a normal case the duration of the vertigo is approximately 24 seconds.

The test is performed in a similar way by turning the patient to the left.

This quantitative test for vertigo is not necessary as a routine procedure. "Past pointing," which we are about to discuss and which is routinely tested, makes this unnecessary. When the patient is turned to the right and stopped, he feels that he is turning

to the left, and for this reason he "past points" to the right. That is, when the patient is stopped after being turned in the chair, he has a subjective sensation of turning in a direction opposite to that in which he was actually turned. After touching the examiner's finger at this time, he is under the impression as he is raising his arm that he is turning away from the finger, and therefore tries to correct for that by moving the arm out to the point where he conceives the finger now to be. Since as a matter of fact he is not moving, the chair being held rigidly still, he points widely past the finger and continues doing so in lessening degree as long as the vertigo lasts. The very presence, therefore, of past pointing is indicative of the presence of vertigo—the past pointing being the objective evidence of his subjective impression of turning.

PAST POINTING AFTER TURNING.

The patient's head is again fixed so that the horizontal canals only are tested, and the chair turned at double the speed of that for nystagmus—that is, the 10 turns are made in 10 seconds instead of 20. The patient is carefully instructed as to what is expected of him, and is told to keep his eyes closed throughout the entire test. The examiner stands in front of the patient with the stop pedal near his right foot. As the chair is turned for the tenth time the pedal is released by the examiner's right foot and the speed of the chair gradually slackened, so that the stop is accomplished without any jarring. The patient's right hand is then quickly grasped, and after his forefinger touches the examiner's finger, the examiner says, "up," upon which the patient raises the arm in question to the perpendicular and immediately tries to come back to the examiner's finger. The "past pointing" is called off in inches and recorded by the assistant where it says "shoulder from above," as, for instance, "15 to R." (15 inches to the right). The left arm is immediately tested in the same way, then the right arm again, then the left arm again, and so on until there is no longer any past pointing. In a normal case the vertigo lasts sufficiently long to permit of three past pointings of each arm, gradually lessening in extent until the patient is again able to find the finger.

The patient is then turned to the left and the past-pointing of both arms taken and recorded as above.

The patient's eyes must be kept closed throughout these tests, and if his cooperation is doubtful it is best to blindfold him.

The turning tests affect only those semicircular canals which are in a horizontal plane while the patient is turned. Should it be desired to test the two sets of vertical canals by turning, the patient's head must be placed either 120° forward or 60° backward. For turning with the head forward, a small bar with a comfortable head-

rest is placed between the two arms of the turning chair and the patient turned ten times, first to the right. The resulting nystagmus and vertigo are, of course, in the frontal plane—a rotary nystagmus to the left and vertigo to the left.

In taking the past pointing the patient keeps his head in the same position after stopping the chair, and the examiner is forced to kneel on the floor in making the test. The past pointing is not so large as that obtained from the horizontal canals, but is with both arms to the right after turning to the right, and with both arms to the left after turning to the left.

When testing the vertical canals with the head back, the upright, or turning, handle is loosened at the ratchet and turned back. The headrest is brought all the way down and the head comfortably fixed in a position 60° backward. The patient is turned to the right, and there is produced a rotary nystagmus to the right (just the opposite to that obtained from turning with the head forward). The past pointing, however, is also to the right, just the same as that obtained after turning to the right with the head upright or with the head forward. It may be remarked in passing that this test does not bear out the statement and law laid down in all the writings on this subject, including Bárány's, that "the past pointing is always in the direction opposite to that of the nystagmus." Nystagmus and vertigo (with its consequent past pointing) are not in any way dependent on each other. They are different reactions produced by stimuli sent along entirely different paths. A normal individual always past points to the right after having turned to the right, regardless of the position of the head. Nystagmus, on the other hand, is in a diametrically opposite direction, after turning with the head backward from what it is on turning with the head forward. Turning to the right with the head back produces a nystagmus to the right and a past pointing to the right, so that the statement that "past pointing in a direction opposite to the nystagmus" is not only misleading and confusing, but actually incorrect.

FALLING AFTER TURNING.

This results when the individual tested has a subjective sensation of turning in a vertical plane. He will fall to the right or left if he thinks he is turning in a frontal plane, and will fall forward or backward if he thinks he is turning in a sagittal plane. Therefore, turning a patient with the head tilted 30 degrees forward produces no falling, as the patient feels he is revolving in a plane parallel to the floor. Similarly, turning with the head forward or backward produces no falling so long as the head is maintained in this position. If, however, after turning with the head forward or backward, the

head is brought to the upright position, the subjective sensation is now one of turning in a plane at right angles to the floor, and the patient falls either to the right or left. Again, if the head is inclined toward the shoulder, after the turning the subjective sensation is one of turning in a plane parallel to the floor; if the head be raised to the upright position, however, the subjective sensation is that of turning in a sagittal plane, and the patient falls forward or backward. These tests, however, are seldom necessary in routine examination.

CALORIC TEST.

The main advantage of the caloric test of Bárány is that it enables us to examine each internal ear separately, and also to analyze the function of its canals separately, whereas turning stimulates both labyrinths at the same time. It is essential to have an absolute standard in the matter of temperature of the water. As Bárány directs, we employ water at 68° F. This temperature is sufficiently cool to secure a good reaction, and yet not cold enough to be uncomfortable to the patient. When hot water is to be used the temperature is 112° F. The latter should be but seldom used.

The vessel containing the water is placed about 2 feet above the level of the ear to be douched. The shape or size of the nozzle is immaterial, the essential thing being that a free and continuous stream of water shall flow against the drum membrane maintaining an even temperature of 68°. The nozzle has a closing valve within easy reach of the fingers. This valve also enables the examiner to regulate the force and volume of the stream of water entering the canal. A rubber receptacle is placed underneath the patient's ear to catch the return flow of the water. A tube at the bottom conducts the water into a basin below. When this is properly attached there is little chance of wetting the patient, and if necessary douching can be kept up for several minutes without any interruption.

Remembering the desirability of testing only one set of canals at a time, and recalling still further that this caloric test influences only those canals which are in a vertical plane, the patient's head is placed 30° forward so as not to include the horizontal canal in the reaction. The chair is held firmly by the foot clamp, the stop watch is held ready, the nozzle is inserted into the canal, and the instant at which douching is started the stop watch is clicked. The patient's eyelid is elevated; the patient is asked to look down and the eyeball carefully watched for any rhythmic nystagmus to appear. The moment that a rotary rhythmic nystagmus appears the stop watch is clicked again and the number of seconds necessary to produce the reaction noted. We usually continue the douching for a few seconds longer in order to obtain the maximum reaction. In the normal, nystagmus appears after 40 seconds, and after douching for 5 or 10 seconds more shows a large amplitude. The direction of the

nystagmus is recorded by a curved arrow on the chart where it says "douche right." The amplitude is noted beneath and then the length of time required to produce it.

The patient is then told to close his eyes and the pointing tests are carried out and recorded in the same way as before described. It is well to remember that this douching has produced vertigo in the frontal plane—the same reaction that occurs after turning with the head forward; the patient, therefore, exhibits a marked tendency to fall, and it is advisable to have an assistant hold the patient's head firmly in the head rest while taking the pointing tests. As soon as the past pointing of the arms has been taken the head is quickly tilted to a position 60° back, which places the horizontal canal in the vertical plane in a position in which it can be affected by the chilling. The patient is told to look upward, and the existing rotary nystagmus immediately becomes horizontal. The pointing of both arms is then quickly taken with the head in that position. The head may then be tilted forward 90° , and the past pointing of both arms be taken again. This new position of the head again influences the horizontal canal, but has reversed the direction of the endolymph movement. All the reactions, therefore, are also reversed. The duration of the average stimulus after douching is long enough to permit of the examination of the head in the three positions just given. It is well to note that in this way one douching of only one ear can test out all the pathways for nystagmus as well as produce past pointing in all directions, enabling us to test out the integrity of the entire cerebellum.

Falling.—The caloric test by its very nature produces vertigo only in a plane at right angles to the floor, so that "falling" always occurs on douching a normal ear. All that is routinely necessary is to observe the tendency to fall while the other examinations are going on, and to note it on the chart.

ELECTRICAL.

For practical purposes the electrical tests have been of only slight use in our experience. Since the ability to examine one set of canals at a time is of such prime importance in making these tests of great clinical usefulness, it is evident how limited are the uses of an agency which stimulates at the same time not only the entire labyrinth, but the eighth nerve as well, and perhaps even the medullary nuclei. It is of great use, however, when the question arises as to a differential diagnosis between a destruction of the labyrinth and the eighth nerve. In a recent destruction of the labyrinth the caloric test will produce no reactions. The electric current, however, may directly affect the eighth nerve and produce normal reactions. When

the galvanic current is used a large electrode is held in one hand, and a small one is placed on the mastoid process. Both electrodes should be covered with cloth or cotton fairly saturated with a normal salt solution. The current is gradually turned on, and when four milliamperes are discharged a nystagmus should appear.

It must not be thought that a routine examination of the internal ear requires all the preceding tests. In order to illustrate the simplicity of a complete average examination of the static labyrinth we insert a chart properly filled in with the results of the examination of a normal individual. Naturally, for example, if douching the vertical canals of both ears gives a doubtful response, it is wise to verify the examination by turning with the head back—or forward; in this case the findings are recorded on an extra chart to avoid confusion. In the average case, however, it is necessary merely to fill out one vestibular page, as shown, and to conduct all the tests with the head upright; the turning in this position tests the horizontal canals, and the douching examines the vertical canals. The examiner needs merely to complete this chart to have before him all the essential data of a routine examination.

PART 4.

ABSTRACTS FROM WAR LITERATURE OF HEAD INJURIES.

ENGLISH SCHOOL.

A survey of the recent literature of the war, as the subjoined extracts show, demonstrates what has been said in the preface, namely, that exigencies of time, place, and circumstance modify the fundamental (we might almost call them orthodox) principles laid down by Cushing. These variations are best appreciated in gross by grouping the literature under the heads of English, French, and German. As might naturally be expected, however, there are variations of opinions even in the ranks of these three schools.

Wilfred Trotter, for many years an enthusiastic worker in the field of neurological surgery, correlates fundamental facts with lessons learned in the war zone:

Trotter, W.: The Principles of the Operative Treatment of Traumatic Cerebral Lesions. *Brit. J. Surg.*, 1915, ii, 520.

The article itself is divided into four sections, with many subdivisions, all of which tend to bring out the correlation referred to above.

Under the heading, "Physiological peculiarities of the cerebral circulation," a short anatomical description of the brain is given, with special stress on the absolute inelasticity of the craniodural capsule and the close application of this capsule to the brain. These facts are used in the sections on encroachments on the intracranial cavity.

1. Of encroachments of vital origin, hemorrhage is the chief. The effects noted are due solely to an interference with the circulation in that part of the brain affected: (1) Stage of compensation: With a developing hematoma, space is afforded for the blood, without impairing cerebral circulation, (*a*) by a displacement of the cerebrospinal fluid, and (*b*) by compression of the regional veins, which allow of a certain amount of compression before congestion. (2) Stage of venous obstruction: As hemorrhage increases, the compression on the veins also increases until they are finally obliterated, causing a congestion and cyanosis of the brain in that region. In many cases of traumatic compression the process goes no further and gives rise merely to an increased excitability of cerebral tissue. (3) Stage of anemia: Further increase in the hematoma leads to a collapse of the capillaries, and a white area is

formed immediately beneath the clot, from which the blood is totally squeezed out. This gives rise to paralytic symptoms, which condition is invariable and characteristic. If the hemorrhage continues, these areas gradually enlarge until more and more brain substance is involved, with corresponding symptoms—the three zones of compression, however, maintaining their entities.

Encroachments due to external violence fall under the heads of (1) deformation of the skull through external violence, as a fall on the head, and (2) traversing of the skull by high-velocity bullet. The physical consequences of both are identical. At the moment of injury there is a very great increase of intracranial tension, and the entire brain is subjected to hyperacute compression. This gives rise to total, but momentary, capillary anemia of the brain, with resultant widespread paralytic symptoms, and is known as concussion of the brain. It is characterized by (a) instantaneous onset; (b) paralytic symptoms referable to all parts of the brain; (c) tendency to spontaneous recovery; and (d) absence at post-mortem of any characteristic findings.

1. In injuries accompanying deformation of the skull, as a rule, there is an inbending of the skull, but no depressed fracture. Part of the force being transmitted throughout the cavity causes concussion and part directly affects the brain substance. The brain is injured (1) at the point of impact of skull and brain, direct contusion; (2) diametrically opposite—contrecoup or polar contusion; and (3) between these two points, in scattered foci—substance contusion. Also the sudden displacement of cerebrospinal fluid in the ventricles may cause foci of contusion through the narrower parts, the Sylvian aqueduct.

2. Injuries due to transit of a bullet depend on the velocity of the bullet. At maximum velocity, the cranial contents acting as continuous homogeneous medium, the skull is shattered, the scalp torn open, and the brain disorganized. At slightly less velocity the scalp remains whole, but the brain and skull are destroyed. As velocity declines, this explosive effect is the first to disappear, except at the exit wound. At low velocities there is intense hyperacute compression of the brain, an explosive effect at the exit wound; but as long as the cranial vault remains intact, extensive destruction of the brain does not occur.

3. Injuries associated with localized fracture are essentially local. The scalp is lacerated, the skull comminuted and depressed, and the brain contused or lacerated locally, with no scattered or distant foci of indirect injury, no polar contusion, and slight concussion. There are three practical rules in these cases: (1) *In adults, invariably all depressed fractures are compound.* (2) *The damage to the brain is often underestimated.* (3) *The prognosis is better than the wound would indicate, because there is no polar contusion or distant lesions.*

There are three modes of action of cerebral injuries as follows:

1. **Direct destructive effects.**—Most commonly by bullets, and easily recognized.

2. **Reactionary swelling.**—Injury of the brain leads to edema and swelling, which in turn leads to pressure on the veins and venous obstruction; but there is no capillary anemia, and the symptoms are chiefly confined to the irritative phenomena. It is when this condition progresses below the tentorium into the vital centers that the gravest effects are produced. In concussion of the brain the irritative symptoms coming on several hours after injury are due to this reactionary edema causing venous obstruction, and a purely decompressive operation is indicated.

3. **Hemorrhage.**—This is the most important mechanism by which symptoms are produced after head injuries. The situation has an important effect on the size of the hematoma, and the rate of bleeding is even more important, considering the symptoms and the gravity of the case.

(a) *Extradural.*—Found chiefly in the middle fossa and usually from the middle meningeal artery; may come from the veins and may be located in the frontal region.

(b) *Subdural.*—Commonest cause of severe compression. In the acute, the hemorrhage readily and rapidly extends over the whole hemisphere, which is displaced toward the opposite side. It may spread to the posterior fossæ and bulbar symptoms occur. The pressure often forces the brain into the tentorial foramen, preventing the spread into the inferior fossa and subsequent bulbar involvement.

(c) *Cortical.*—Arises from the vessels of the cortex and is the most common cause of Jacksonian epilepsy. It is the result of cerebral contusion.

(d) *Intracerebral.*—Is very uncommon and very grave. Is due to severe cerebral contusion from one of the distant foci of injury in the brain substance, is accompanied by other contusions, and usually without fracture of the skull.

(e) *Intraventricular.*—Is usually an extension of the intracerebral into the ventricle and is very uncommon.

Clinical types of cerebral injury are as follows:

1. **Grave and extensive hemispherical compression.**—After severe injury concussion develops, which passes off, followed by a lucid interval, and then a comatose state, with hemispheric signs. Progressing pupillary changes occur and, later, irritative bulbar symptoms. Operation is indicated, the opening to be made in the temporal fossa and to be small. If the brain bulges strongly, a decompression is necessary. The dura should always be incised to discover a concealed hematoma and it should not be sutured afterwards. Drainage usually is not necessary.

2. **Grave injury without localizing signs.**—After severe injury with fracture of the base and external hemorrhages, concussion occurs, and shades into more or less complete coma without a distinct interval. Usually this coma is not

profound. There is some rigidity of the limbs; reflexes are exalted or depressed; there is no definite pupillary change and no incomplete bulbar signs. Such a case, if of severe grade, is usually fatal, but milder conditions often recover spontaneously. They are prone to pneumonia and meningitis, however. No direct treatment is indicated, because there is no definite focal lesion. If coma becomes profound and persistent for days or weeks, that in itself localizes the pressure in the superior chamber and indicates a temporal decompression operation. The results are usually satisfactory.

3. Cases with mainly irritative signs.—(a) *Severe head injuries with mental excitement.* From a state of concussion the case passes into a violent, uncontrollable delirium, as if fighting drunk. A diagnosis is often impossible immediately, and usually only after observation. Generally this delirium is more impenetrable to external influences and signs of severe headache are present. As a rule, it is necessary to delay operation until some definite paralytic phenomenon appears.

(b) *Classical "cerebral irritation."* After concussion has passed, a typical state of cerebral irritation supervenes. The case is very irritable, pulse usually quick, temperature raised, and always a severe headache. There is no mental confusion, but no spontaneous effort, mental or physical. Occasionally, however, there is delirium, especially at night. This is a condition of moderate venous congestion with edema, caused by multiple foci of contusion through brain substance. Spontaneous recovery is usual, but is generally followed by headache, giddiness, loss of memory, and even epilepsy. If severe, temporal decompression is indicated.

4. Cases with signs of localized lesion.—These are mainly cases of cortical hemorrhage after direct or polar contusion. Symptoms of localized cerebral irritation usually show after several days or a week, and are most common in the motor area. Operation should be performed as soon as localization of the hematoma can be made.

5. Compound depressed fracture.—There is a remarkable absence of symptoms of concussion or distant injury, and the prognosis is surprisingly good. The diagnosis is usually easy, but a skiagram should always be taken. Unless the injury has directly affected some part, there will, as a rule, be no primary cerebral symptoms. All cases demand operation, whether they display cerebral symptoms or not. The chief object is to limit sepsis, remove foreign matter, and provide drainage, but extensive explorations into brain substance must not be done.

Treatment.—*In all other classes of head injury Trotter repeatedly insists that the treatment must be based upon evidence of cerebral injury; that is to say, the surgeon must regulate his operative interference solely upon the evidence collected by systematic neurological examination of the patient. This principle does not apply to the type now under consideration. All of these cases demand primary surgical*

interference, whether they display cerebral symptoms or not. The chief object of such treatment is the limitation of sepsis, the removal of fragments of bone and other foreign substances from the brain, and occasionally the provision for the escape of collections of blood and disorganized tissue.

It will be seen also that this treatment differs from that which has been found most satisfactory in gunshot fractures of the extremities. In such cases it is generally acknowledged that any primary operative interference with the fractured bone is to be avoided, for it is found that the soft parts may be seriously infected without the bone being also involved. An operation on the fracture, therefore, conveys infective material to the previously unaffected bone. In gunshot injuries of the skull the fracture is in close relation to the surface wound and can scarcely fail to participate in its infections. There are, moreover, two especial reasons why such cases should not be treated by purely expectant methods: First, the great danger that spreading infections of the brain and meninges may start from the collection of pulped brain, blood, fragments of bone, and foreign substances which is apt to be retained in the wound; and, secondly, that grave interference with the function of the brain is likely to result from the retention of such materials, even if healing should occur without suppuration.

The surgeon must keep in mind the importance of doing no further damage and of avoiding any risk of actually spreading sepsis. The primary removal of foreign substances deeply embedded in the brain is not always an indispensable necessity, and the satisfaction of recovering a bullet should not be allowed to tempt the surgeon into enterprising explorations at this early period.

If the patient is seen soon after the injury the following treatment should be carried out as far as the circumstances allow. An anesthetic should be given and the scalp about the wound should be shaved and cleansed. It may be necessary to enlarge the skin wound to permit of the proper inspection of the damaged parts. Depressed and loose fragments of bone and foreign substances such as are accessible to gentle and cautious examination should be removed. Any badly damaged and befouled skin should be cut away and the wound left widely open. The whole of these manipulations may with advantage be carried out under continuous irrigation with sublimate lotion (1-1000 to 1-2000).

The wounds of entry and exit due to the transit of a bullet through the skull must be regarded as coming within the class of localized injuries here considered and be dealt with accordingly. Many of such cases, however, will show evidence of gross intracranial hemorrhage, and of course urgently demand surgical treatment under that category as well.

Should the patient not come under treatment until some days after the injury, infective conditions may already have developed. Nevertheless the same treatment as that already

described must be carried out, whether cerebral symptoms have appeared or not.

The wound in all cases should be treated by large wet dressings frequently changed. Whether or not these are impregnated with antiseptic substances seems to matter much less than whether or not they are kept moist and renewed often. The familiar boracic fomentation, which when carefully prepared and properly applied may be regarded as a typical wet aseptic dressing, is perhaps as satisfactory as any.

It will be noted in the above abstract that Trotter admits in his treatment few of the qualifying limitations incident to war. In this particular, as we shall see later, he differs from most of his English colleagues.

The papers by Sargent and Holmes illustrate admirably this difference in personal viewpoint, and in addition furnish opinions on practically every moot point concerned in the interpretation and treatment of head injuries.

Sargeant, P., and Holmes, G.: Report of Late Results of Gunshot Wounds of the Head. *J. Roy. Army M. Corps*, 1916, xxvii, Sept.

Late results in head wounds are always of interest since they are largely dependent upon the mode of early treatment.

The authors had ample opportunity to study their cases, many of whom were injured 18 months and two years ago. They had authority from Sir Alfred Keough, D. G., to visit all the hospitals in London and vicinity. This gave them an opportunity to study the condition of 1,239 patients. After excluding scalp wounds without any bony or cerebral injury, and where the nature of the injury was uncertain, as well as others in which the information was too recent for a study of late cases, the accurate data is given concerning 610 patients, 75 per cent of whom were studied three months after the infliction of injury. The cases included in the list are more severe than the average of cases diagnosed as gunshot wounds of the head.

The mortality after evacuating to England was as follows: Of the 1,239 cases studied in eight hospitals, the mortality was 3.7 per cent. Some of the cases were severe and died within two weeks after admission. But five cases succumbed after three months, the other deaths occurred before the end of this time. The immediate cause of death could not be ascertained in a considerable number of the cases. In 22 post-mortem examinations, it was found that nearly all had died of the spread of septic infection. In one remarkable case, the bullet had passed through the right frontal region, the base of the brain, then ricocheted off the Pitres portion of the temporal bone, thence through the third ventricle and the posterior third of the corpus callosum and into the left occipital lobe. The patient died at the end of three and one-half months very suddenly, when all cerebral symptoms had

disappeared, as a result of rupture of an aneurism of the posterior communicating artery.

Eleven patients died after operation: Two after excision of cerebral herniæ, two after primary operations, one after an attempt to remove a lodged shrapnel ball deeply embedded in brain tissue, and in the remainder death followed operations to relieve herniæ or to evacuate cerebral abscess. In 10 out of 17 other cases studied at post-mortem, meningitis, and cerebral abscess caused death of the 7 others.

No death occurred in the cases studied when the dura remained uninjured by the missile or at operation.

The improvement of physical disabilities with the lapse of time is spoken of in a most encouraging way. The amount of disability is, of course, dependent upon the severity of the injury and the location of the lesion. The authors hold to the view that most of the paralyses, sensory and visual disturbances, etc., noted in the earlier stages are due not so much to the result of destruction of brain tissue as to concussion, edema, and vascular disturbances that extend beyond the primary injury, and here they might have added as a result of the vibratory force incident to high velocity on the part of the projectile. The temporary nature of paralyses in the cord is due to the same cause, and here we have noted that the symptoms subside early, just as they are known to pass away with time in many cases of brain injury.

Disappearance of symptoms directly due to destruction of brain tissue are more persistent, and yet the amount of improvement in some of the cases was surprising. A small proportion of cases with perforating and penetrating wounds of the skull afflicted with paralysis, sensory disturbances, hemianopia, etc., have already been returned to active service and others have returned to wage-earnings in vocations in civil life.

Amelioration has been especially noticeable in many of the cases of various forms of paralysis due to injury of the superior longitudinal sinus. As to neurological complications, the authors note that but few cases of insanity or epilepsy have developed among the convalescent patients or those who have been restored to duty. Dullness, loss of memory, irritability, and childishness are oftentimes present in the earlier stages, but these tend to disappear or diminish with time. Only eight cases of insanity were noted in the first 12 months.

As evidence of the apparent rarity of insanity after head injuries, it is pointed out that only one case was received at the London County Council Area, where all cases of insanity in invalided soldiers naturally go, from at least one-seventh of the population of the United Kingdom. Maj. F. W. Mott, of the London County Council Asylums, states that he is very skeptical of a large number of cases of insanity arising from traumatic causes. He believes that head injuries apart from syphilis, alcohol, and hereditary neuropathic taint seldom cause mental affection.

Likewise epilepsy of the Jacksonian type has been surprisingly rare, and, seen in later stages, it has been less common than was feared from the generally accepted opinions on the subject. It has occurred in 6 per cent of the 610 cases. In 8 one convulsion had taken place; in 12 only a few; 5 men were reported to have had 5 or 6 convulsions; while in 11 the convulsions were frequent.

The administration of bromide in all serious cranial injuries until the wound is healed and for some months afterward is considered advisable. Headache is mentioned among other neurological complications; and again a feeling of weight, pressure, or throbbing in the head, aggravated by noise, fatigue, exertion, or emotion, attacks of dizziness and nervous or deficient control over emotions or feelings are noted. Many of the patients exhibit a considerable change in temperament. They become depressed, moody, irritable, or emotional, and unable to concentrate their attention on any physical or intellectual work. These symptoms are neurasthenic in type. They are independent of the site or severity of the original wound and they are often as severe when a minor injury like a scalp wound has been received as in the case of a serious compound fracture of the skull, and they seem to develop just as often after an operation as not.

In reviewing these remote effects of head wounds it is well to remember that a great deal of our knowledge of this class of cases has been handed down to us by medicomilitary writers who had viewed the subject from a military standpoint after years of experience.

Longmore, from his extensive experience, states that few cases of head injury from gunshot, be they contusion or fracture, fail to give symptoms of cerebral disturbance. The fact that paralytic symptoms are more severe at first and tend to disappear wholly or partly has been a matter of common observation by military surgeons. Dizziness, irritability, headache, and other of the neurological symptoms are prone to recur while on duty in hot climates, so that soldiers frequently have to be discharged from the service on this account. Although many of the cases of head wounds have been restored to the colors, it is doubtful if they could continue on duty in tropical countries.

Out of 610 head cases to reach England, 120 had cerebral hernia. The progress and outcome in cases with this unfortunate complication are dealt with according to the different types of wounds. Of the 610 cases 96 had missiles lodged in the brain, and of 26 from whom the missile had been removed by operation 6 developed hernia cerebri, with 2 deaths. Out of 69 cases with missiles in situ 14 developed hernia cerebri, with 2 deaths. Of the 16 who survived with hernia, the hernia had shrunk and the wound had healed when last heard from, and in 2 the herniæ were smaller and the wounds were healing rapidly three and four months, respectively, after the date of injury.

In 68 cases of "through-and-through" shots 14 developed cerebral hernia; 4 out of the 14 died, and in 8 of the others the wounds were completely healed when last heard from. Out of 310 cases of penetrating wounds without retained missile, 86 reached England with cerebral hernia. There were 19 deaths among these, and in 49 the wounds healed completely. Of the 86, 62 had been operated upon abroad.

Mention is made of 69 cases of lodged missile in the brain at some distance from the point of entry. A few of the missiles were rifle bullets and a few were shrapnel balls, but the great majority were fragments of shell, frequently multiple and not accessible. Four of the 69 cases died, and of the surviving 65 cases, 12 had been wounded less than 3 months previously, 25 between 3 and 6 months, 21 between 6 and 12 months, and 7 for over 1 year. In 76 per cent of the cases the wounds had completely healed; in 30 per cent complete recovery had occurred and no symptoms of cerebral lesion were present; in 40 per cent the neurological symptoms had improved to a remarkable extent; in 10.5 per cent hemiplegia, hemianopia, neurasthenia, etc., still persisted, but the condition is attributable to the damage done by the missile rather than to its presence.

The conclusions are as follows:

The later results of head wounds as shown by their report is more satisfactory than had been generally expected. It is observed that the proportion of patients who die after transference to England (fixed hospitals) is small, but the same is true of all war wounds of the head.

Later complications, such as cerebral abscess, are relatively rare, and later complications or sequelæ, as insanity and epilepsy, are as yet much less common than has been foretold. The diminution of cerebral hernia is doubtless due to antiseptic treatment of the wounds in the beginning, and as for the later sequelæ more time than one year should be allowed to pass before a definite statement can be made as to their possible occurrence.

The authors recommend only such surgical intervention at the front as may be called for to establish necessary drainage and the healing of the wound. Every precaution should be taken to prevent the development of hernia cerebri. The advice to avoid the attempt at removal of lodged foreign bodies in the brain is sound, since it may involve spread of infection or further destruction of brain tissue, but to state that many patients with foreign bodies lodged deeply in the brain are scarcely more liable to serious complications than men in whom the brain had been merely exposed and lacerated sounds premature in view of the short time which has elapsed since the occurrence of injury and the lack of opportunity to study the cases under the varying and trying conditions of vocations generally.

Sargent, P., and Holmes G.: The Treatment of Penetrating Wounds of the Skull. *Brit. Jour. of Surg.*, Vol. II, 1916, p. 474.

Skull wounds are classified as follows:

A.—Tangential wounds in which the missile, usually a rifle bullet, has passed superficially to the bone, leaving its mark externally in the form of a gutter of the scalp, or of two wounds with a bridge of skin between them. Tangential wounds of this kind may cause no fracture at all, or merely a slight depression of the inner table without laceration of the dura mater. In the more severe cases, however, the bone is comminuted, and the dura lacerated, whilst at the same time fragments of bone are driven into the brain substance, though rarely to any great depth.

B.—Penetrating wounds in which the missile has passed immediately beneath the bone, along the chord of a small arc. In these cases laceration of the dura and brain is naturally inevitable; and although the bony fragments are for the most part driven outwards, yet some may at the same time have penetrated more or less deeply. These wounds often result in the destruction of an area of cerebral cortex which is relatively large in extent as compared with its depth, presenting a condition for which some operation of covering-in the exposed brain, combined with free drainage, may be adopted.

C.—Direct localized blows by fragments of shell or shrapnel bullets which have themselves failed to penetrate the skull, but which nevertheless often drive fragments of bone deeply into the brain, so deeply in fact that even if the ventricle is not actually opened, its proximity to the septic track is such as to render its infection highly probable. In many of the fatal cases post-mortem examination has shown that this condition existed. In removing fragments of bone from any depth, the position of the lateral ventricle should always be kept clearly in mind, lest it should be accidentally opened in the course of the operation.

D.—Penetrating wounds in which a missile has entered, and remained lodged within the brain substance.

E.—Cases in which a rifle bullet has passed across the cranial cavity, the wounds of entry and exit being on opposite sides of the head, or so far apart that the intervening bone is undamaged, or at most only fissured. Here it is only at the entrance wound that fragments of bone are in-driven; and as the bony opening is usually only a small puncture, the fragments are often mere splinters.

Loss of consciousness of varying depth and duration, general muscular flaccidity, and disturbance of cardiac, respiratory, and vasomotor action, are amongst the most striking of these general symptoms; but their exact pathological or mechanical basis is still imperfectly understood. Whatever it may be, it results in a suspension or disturbance of function from which (except in patients who die outright or within a few hours) recovery can take place spontaneously.

Associated with these disturbances we find a group of symptoms due to the abnormal increase of the intracranial

pressure. This is the stage of cerebral œdema, and its effects may be aggravated by the presence of effused blood, which either mingles freely with the excessive cerebrospinal fluid or may be more or less localized or circumscribed. The experience of a large number of cases has shown that such hemorrhages are very rarely large enough either to threaten life or to demand operative interference. The possibility of a hemorrhage sufficiently large to demand, and sufficiently localized and accessible to be amenable to, direct operative treatment, is therefore so removed that exploration on this account is rarely desirable, more especially in view of the danger of infection which would be incurred from the presence of a septic scalp wound. The stage of cerebral œdema may last for several days, and in the absence of a spreading infection, subsides spontaneously.

In addition to the symptoms of general cerebral disturbance of function, those due to the local damage must be taken into account. Evidence of the local injury is afforded by disturbance of motor, sensory, reflex, mental, and visual functions, according to the region involved. Such impairment of function depends partly upon *actual destruction* of cerebral tissue, and partly upon *contusion, localized œdema, local "concussion"*; *from the effects of the former no improvement can be obtained by operation, while that part of the symptom-syndrome due to the latter causes is capable of more or less complete recovery without surgical interference.* As it is not possible at first to say how great a share the one or the other contributes to the clinical picture, no accurate prognosis can be made. *It is certain, however, that operative interference can not directly benefit either condition, and recovery of function is sometimes wrongly ascribed to the operation which precedes it.* On the other hand, any progressive loss of function usually points to the necessity for affording free drainage of the damaged brain, as it may be due to a secondary inflammatory process which may lead to an extension of the area permanently damaged.

In many respects all these penetrating injuries are alike; they present similar difficulties, are attended by the same danger, and offer similar problems. It is easy to regard them merely as compound fractures complicated by injury to underlying structures, and to assume that they require exactly the same treatment as compound fractures in general. This view holds good so far as the essential point of eliminating infection is concerned, but the problem is by no means so simple as at first sight it appears to be. How to secure efficient drainage of the damaged brain is one of the chief difficulties, and this arises principally from two facts; first, that the brain swells so easily in response both to traumatism and to inflammation; second, that the amount of extra room which the cranial cavity is capable of affording is so small in relation to this swelling.

As regards the operative details, these cases may be looked upon as falling into one of two groups, according to the

depth to which bone fragments have been in-driven and to which brain tissue has been damaged. In the first and larger group are found those cases in which the scalp wound, bone defect, and dural opening are comparatively small, but in which the underlying brain has been penetrated and lacerated to such a depth that, after the removal of bone fragments, the tract requires drainage by a tube. In the second are included those in which the dural laceration and the cerebral injury are greater in extent than in the former group, but are comparatively superficial—where, in fact, after cleaning and removal of bone fragments no tract into the brain exists into which a tube could be inserted.

The time at which operation should be undertaken is a matter of very great importance and one upon which opinions vary. In considering the question, it is necessary to have a clear conception as to what ends are to be attained by the operation and in what manner it is expected to benefit the patient. Two main considerations must be taken into account, namely, the cerebral injury as such, and the presence of an infected wound. As regards the neurological symptoms, operation is rarely if ever called for at an early stage, and seldom at a later one. From the point of view of the wound itself, it would naturally seem at first sight that immediate operation would offer the best prospect of recovery and prompt healing; and this would doubtless be true but for two special circumstances: Firstly, the ease with which the subarachnoid space can be infected; secondly, the tendency which exists toward the formation of a hernia cerebri. Experience has shown that delay minimizes both these dangers, as may be appreciated from the following considerations:

Firstly, as regards meningitis: The chief safeguard against a generalized meningeal infection is the formation of adhesions between the edges of the dural wound and the contiguous pia-arachnoid, by which means the subarachnoid space is shut off; this process is assisted by the pressure of the swollen brain against the dural opening.

Secondly, as regards hernia cerebri: When the intracranial pressure is raised, and there exists a defect in skull and dura mater, brain matter tends to be extruded. In the early or concussion stage the brain is swollen from traumatic edema, and consequently the intracranial tension is raised considerably above the normal; any fresh manipulation is followed by a further rise in pressure; with any meningeal infection the pressure is still further increased. The formation of a hernia cerebri is not only dangerous to life, but also to function—the function not only of the brain actually protruded, but also of that part which lies in the neighborhood of its base. This danger, then, provides a second reason against immediate operation. Naturally there is some risk in allowing bone fragments, which may be infected, to remain buried in the brain, because an infective encephalitis may spread from such a focus and may reach the ventricle. The respective dangers, those of meningitis and of hernia formation

attendant upon the earlier operation and that of ventricular infection which besets delay, have to be balanced against one another; but the experience of a large number of cases has gone to show that the dangers of the earlier operation are the greater.

The question of the removal of metallic fragments is difficult to decide, inasmuch as their ultimate fate and their possible effects upon the surrounding brain are at present uncertain. Such evidence as has accumulated up to the present time seems to show that foreign bodies are best left alone unless they are so superficial as to be easily removed along with the bone fragments; or unless at a later period they cause symptoms directly referable to their presence.

Two considerations make one hesitate before removing even easily accessible pieces of metal. Firstly, although several patients have made good recoveries after removal, several others have died, undoubtedly as the direct result of the operation. Secondly, many patients in whom removal was not attempted have gone home with good prospects of recovery, and several of these of whom we have since heard are still, after many months, doing well and with the wounds completely healed.

If it is decided not to remove the foreign body, the wound of entrance must nevertheless be dealt with as efficiently, and upon the same lines, as if no foreign body were present.

The authors say that the following operation has furnished them their best results:

The whole scalp is cleanly shaved, thoroughly rubbed over with gauze dipped in alcohol, and painted with 2 per cent iodine spirit; the wound shares the same treatment. When the position of the operative field is convenient, a tourniquet of rubber drainage tubing is tied tightly round the head below theinion, and immediately above the auricles and eyebrows; to prevent the band slipping down over the brows, and possibly damaging the eyeballs, it is secured to the scalp by a couple of sharp-pointed skin clips. *The operation is conducted from start to finish under a rapid stream of hot normal saline solution.* This is an important point, as much softened brain and infective material is washed away, and at the same time the operative field is kept unobscured by blood.

The next step is to excise the edges of the scalp wound just above the limits of visible bruising and laceration of the skin; any torn and bruised aponeurosis or muscle can be cut away from beneath after turning down the flap. A large flap is now cut, the shape and size of which necessarily vary with the position and size of the wound which occupies its center. It should be large enough to expose an area of bone well beyond the limits of the bony opening which is to be made; *it is better to make too large than too small a flap.*

The opening in the bone is now enlarged so as to expose the dura mater for at least half an inch all around the dural wound. Usually it is best to make a trephine hold alongside the bony opening, and to work from this; if one at-

tempts to enlarge the existing opening by inserting a forceps blade beneath its edge, there is considerable risk of producing further damage and of disturbing the adhesions between dura and pia-arachnoid at the edges of the dural wound. Any loose fragments of bone are next picked out, and then the finger is very gently inserted through the dural wound into the softened disintegrated brain. Directly a bony fragment is felt it is picked out with dissecting forceps. This procedure is persisted in until all the fragments have been withdrawn. *It is of the utmost importance not to enlarge the dural opening, which is almost invariably of sufficient size to permit of the necessary manipulations.* At this stage it almost always happens that, with the escape of softened brain and the removal of the bony fragments, the general intracranial tension is less than at the beginning; the dura pulsates more freely, and further escape of particles of brain and bloodclot takes place. This cleansing is assisted by directing the stream of hot saline upon the opening, a proceeding which at the same time checks and arrests any bleeding that may be present.

The track having thus been cleansed as thoroughly and at the same time as gently as possible now requires drainage. The tube which we have found, after several experiments, to answer the purpose best is a cylinder of perforated metal (zinc, aluminum, or copper) $1\frac{1}{4}$ inches long and three-eighths inch in diameter. This tube is very carefully inserted into the track, and the flap is replaced and sutured, a small drain of rolled rubber sheeting being inserted between two stitches at each inferior angle. By the time that the flap suture is completed it is usually found that a certain amount of disintegrated brain has already been squeezed into the tube through the lateral holes. This material is removed by means of a small curette, still under the saline stream. The lumen is now dried out with gauze strips and filled with sterilized glycerin; finally, a packing of gauze soaked in glycerin is placed around the tube and a large dressing of gauze and wool is applied. Glycerin has proved to be the best application; it is hypertonic and consequently appears to act in a manner similar to that of a hypertonic salt solution; it has an inhibitory action upon the growth of pyogenic cocci and it seems to emulsify and to facilitate the escape of the disintegrated brain substance. If the intracranial pressure is so high that the tube tends to be extruded a little cerebrospinal fluid is withdrawn by lumbar puncture, and this process can be repeated from day to day, or as often as the degree of intracranial tension requires it. At the subsequent daily dressing the contents of the tube are removed by curetting and syringing; the interior is then dried out and again filled with glycerin. The tube is allowed to remain in for about a week, but at each dressing it must be gently rotated lest it become fixed by granulations or brain matter projecting through any of the holes in its wall. It is not extruded unless the intracranial pressure is unduly raised, and thus any tendency to extrusion is to be regarded as an indication for lumbar puncture.

Removal of shell fragments and bullets.—In a certain number of cases we have considered it advisable to remove these foreign bodies, especially when they lay in comparatively superficial and accessible positions. It is, of course, necessary to recognize the fact that such operations entail a certain amount of additional damage to the brain, as well as the risk of spreading infection. *It is therefore essential that the utmost gentleness should be used* to avoid these accidents as far as possible. We have found that in the case of magnetic metal—namely, fragments of shell and German bullets, which together constitute by far the largest number of such foreign bodies—the use of a giant magnet has enabled us to effect the removal with a smaller amount of manipulation and damage than is possible when scoops and forceps have to be employed.

When the brain has been traumatized superficially and it is not necessary to use a drain, the brain surface is covered over by plastic flaps fashioned from the peri cranium lining the skin flap.

Lumbar puncture.—*Our experience has shown that lumbar puncture is one of the most valuable aids which we possess in the treatment of gunshot wounds of the head.*

Firstly, it may be made use of to diminish the intracranial pressure in the stage of traumatic edema when there is any tendency to the formation of a cerebral hernia, and whilst waiting for the sealing off of the subarachnoid space, which, as has been already explained, renders operation more safe. If employed during this stage, it must be done with great caution, as certain definite dangers attend the removal of too large a quantity of cerebrospinal fluid.

In the second place, lumbar puncture is made use of for controlling any tendency to hernia formation after operation. If the pressure rises so that the tube tends to be extruded, a lumbar puncture should be done; the tube, in fact, affords a valuable indication as to the degree of intracranial pressure, and therefore as to the advisability of performing lumbar puncture.

Whittaker draws attention to the two tremendously important factors of rapid operative technique and very free decompression. He is strongly inclined to agree with the previous authors in his advocacy of delaying operation.

R. Whittaker: Gunshot Wounds of the Cranium, With Special Reference to Those of the Brain. *Brit. Jour. of Surg.*, Vol. II, 1916, p. 710.

One of the first things Whittaker learned in connection with these cases was *that the length of time taken over the operations was of paramount importance.* Most of the patients were desperately ill on arrival and prolonged manipulations of any sort, no matter how desirable in theory, were in practice foredoomed to a fatal termination.

Throughout these operations the dominant note must be *speed—speed and free decompression. The brain itself is able to deal with small foreign bodies left in its substance, provided always that it be given room through which to do so.*

It has been pointed out that possibly these statements, through misinterpretation, might be regarded as dangerous. In theory it is essential to remove every atom of foreign matter in the brain, and every portion of the surrounding brain substance which has been in contact with and become infected by these fragments if the patient is to have his best chance of complete recovery. Nevertheless, from purely practical considerations, the method of treatment described above is best. The following conditions are necessary for the complete and safe removal of all foreign bodies: Perfect X-ray pictures; a correct interpretation of them; a knowledge of the nature and extent of the infection; such a condition of the patient as will permit of the delay required by these observations; *and a perfect equipment and environment.* It is rare for even the majority of these conditions to be obtained; and in the present series, although we had an excellent equipment and environment, yet the worst cases came in in groups of several at a time, together with other serious cases which required attention; X-ray pictures were seldom available within the first few hours of admission; and many of our cases presented signs of such urgent gravity that nothing justified delay in operation.

From a practical point of view it is better to act as suggested; to get the patient safely off the operation table and through a convalescence which is stormy and anxious—watching, it may be, the extrusion of several pieces of bone from his cerebral hernia, and finally, to wish well as he starts for home—rather than to do an operation which the pathologist, noting that no bone, metal, or hair has been found by him in the brain at the autopsy can certify was complete.

After excision of the septic wound or wounds, suitable incisions are made and scalp flaps turned down to expose the area of skull to be dealt with on a scale which allows for the fact that in almost every case more damage is found and more has to be done than was anticipated.

From the moment the operation is started the whole wound area is kept under continuous irrigation with 1–40 carbolic (temperature 110° F.).

Bleeding vessels in the brain or dura and the great sinuses when wounded are dealt with by muscle grafting. The control of hemorrhage from a big sinus, when done by gauze packing, is a never-ending source of anxiety and annoyance. A piece of muscle or fascia taken from the wound, held by an assistant over the bleeding vessel, with the intervention of a smooth rubber glove or a dam for a very short time, while the operation is continued, will control any hemorrhage from the brain or its coverings.

Make an adequate opening in the skull, after which obvious foreign bodies, etc., are lifted or washed out. Any blood clot (and there is usually a surprising amount of this) is scraped or washed away, unperforated rubber sheet or dam is put over the exposed dura mater and brain, and a big perforated rubber sheet over the wound area. The original operation flaps are turned down over this, and the wound is dressed with 1-20 carbolic on cyanide gauze. Half a grain of morphine is then given, and the patient returned to bed. As much more morphine as may be necessary to keep the man quiet for 48 hours is given as occasion arises.

The only other drugs employed are (1) urotropine in 10-grain doses, given every six hours from the first in every case; (2) aspirin and trional together, 10 grains each, as frequently as may be necessary to control the heartache which is often present in the earlier stages. These drugs are remarkably efficacious in their action.

The effects of lumbar puncture are very transitory, lasting a few hours at the most. Its performance can not fail to mean an added strain to a case that has already as much as it can bear. It is better to trust to free decompression for the relief of intracranial pressure. The cases in which the lumbar puncture is likely to be of any use are comparatively few, and consist of those with meningitis, and not those with increased intracranial pressure due to other causes.

A. *The following conditions without doubt demand immediate operation:* (1) Active septic processes in a badly drained wound; (2) evidence of cerebral irritation, as fits, restlessness, or delirium; (3) evidence of cerebral compression, notably severe headache; (4) coma and slow pulse.

B. *Cases in which operation is demanded by the conditions, but should if possible be deferred.*—This group consists essentially of those with active and acute sepsis of the scalp associated with evidences of cerebral compression or irritation. In these cases, before opening the skull, it is necessary first to clean and drain the superficial tissues, a result which can usually be accomplished within 24 hours, by free incisions and boracic fomentations changed hourly, with the application of hydrogen peroxide on each occasion.

C. *Cases requiring an operation which may be postponed according to convenience.*—Clean cases, in which the X rays demonstrate depressed fragments of bone; and those with foreign bodies of moderate size, which can be localized definitely and are in positions which can be reached without further injury to the brain.

D. *Cases in which no operation is required.*—These are as follows: (1) Those with no evidence of sepsis, bone displacement, or foreign body; (2) those with no evidence of sepsis, but with a foreign body not causing progressive symptoms; (3) cases already submitted to a primary operation, in which both free drainage and efficient decompression have been provided.

E. Cases about which there must always be some doubt as to operation.—(1) Those patients whose general and local conditions are apparently hopeless; (2) cases already operated upon, in which the wound is healed but the general condition remains unsatisfactory; (3) cases with healed wounds and no local evidence of sepsis, but with a foreign body, in a position which can only be reached with difficulty, and with symptoms of grave cerebral injury but not of cerebral compression.

On the period elapsing after injury before the operation is performed.—The common idea is that the time which has elapsed between the infliction of the wound, and the patient's arrival in hospital is a dominant factor in the prognosis. But this view is certainly not borne out by the facts here presented. Statistical tables show a mortality of over 50 per cent in the cases which had been operated on before arrival, and a mortality of under 10 per cent in those where primary operation was held over until their arrival at this hospital. This was certainly not due to the selection of the worst cases for early operation on board ship. In fact, the reverse was the case, and in almost all those which had been operated upon before arrival, the nature of the injury had been a small and simple gutter fracture, whilst the bad perforating wounds with sever cerebral symptoms had been left alone.

Roberts, Gray, and Horsely take the stand that delay is fatal to a degree that warrants early (and according to Gray) complete operative interference. Sir Victor Horsley's work is particularly interesting in that it furnishes newer experimental data on the hydrostatic and hydrodynamic effects of high velocity bullets on the skull and brain.

Horsley, V.: Gunshot Wounds of the Head. *Lancet*, Lond., 1915, clxxxviii, 359.

The author has employed modeling clay in carrying out a series of experiments upon the effects produced by high-velocity bullets. The modeling clay resembles the tissues somewhat, in that it contains a considerable percentage of water in its interstices.

The experiments showed that the so-called explosive effect of a high-velocity bullet is directly proportional (1) to the sectional area of the bullet, (2) to the velocity, (3) to the amount of water present in the substance through which the bullet passes, and (4) that the forces of disruption are at an angle to the axis of the flight of the bullet.

Further experiments were undertaken to show (1) where in the course of the bullet the most mischief is done, and (2) by what force. The clay showed that the maximal disturbance is produced as soon as the bullet at its highest velocity is surrounded by the largest mass of wet tissue. This would

explain the larger aperture of exit as compared with the aperture of entrance.

In regard to the forces producing the injury, these relate to the two movements of the bullet: (1) Its progression forward; (2) its spin around a central axis given to it by the rifling. The more important movement from the pathological standpoint is the rotary spin. As regards the influence of the shape of the bullet, the author believes it depends entirely upon the transverse area of the bullet.

Experiments were performed to determine the frequency of the turning over of the bullets. These experiments indicate that bullets turn not infrequently, but turn over only once.

From the clinical standpoint there are several conditions to be considered. Concussion is common and may be fatal without penetration of the skull. Death is probably due to a sudden increase in the intracranial tension, so as to interfere with functional activities of the vital centers.

Rise of intracranial pressure is often due to intracranial hemorrhage, and immediate operation is the only hope for the patient.

Sepsis is a common sequel of head injuries and is frequently due to foreign substances being carried deep into the cranial cavity. Rigid antiseptic treatment is advocated to prevent the occurrence of sepsis. Hernia cerebri may occur either from aseptic or septic wounds.

Functional disturbances of the brain may involve either the sensory or motor areas, and complete restoration of function in these cases is questionable.

Roberts, J. E. H.: The Treatment of Gunshot Wounds of the Head with Special Reference to Apparently Minor Injuries. *Brit. M. J.*, 1915, II, 498.

According to Capt. Roberts there is a large number of gunshot wounds of the scalp to be found in the base hospitals. We might add that this is true of modern wars because the head is exposed to fire more than the remainder of the body as a result of fighting under cover. We may also add that this is specially true of the trench fighting which is now taking place on the western front. Head cases generally form a larger ratio than they did in the days when much of the fighting was done in the open. At the front the surgeon sees all the head cases. The severe cases nearly all succumb in the first 24 hours, and the proportion of cases transferred to the base hospitals is made up of lighter cases, a fairly large number of which come under the class of apparently minor injuries referred to.

In the examination a skiagram is valuable in indicating the presence and location of metallic foreign bodies. It should be taken at right angles to the wound. The findings are not always reliable in indicating fracture. Fracture of

the inner table with depression may be present without showing on the plate, and again the plate may apparently show a depressed fracture of the inner table when no such lesion is present. The wound should not be probed lest superficial infection be carried deeper. In septic wounds and oedematous scalp, unless the indications are urgent, apply hypertonic saline treatment for one or two days before operating. Give urotropine, 20 grains, three times per day in all head cases; shave the scalp completely and paint with iodine.

The operative technique consists in the removal of all infected tissues by steadying the scalp with the fingers and making two incisions surrounding the wound which shall go down to and include the pericranium. The tissue thus isolated is entirely stripped off the bone and removed. The instruments are then rejected. The incision just made is the first step and should not be deferred to a later stage in the operation. The wound should under no condition be enlarged through its septic edges, and never until the completion of the preliminary excision, otherwise the chance for primary union is improbable. The bone is next carefully examined, the periosteum being further stripped up if necessary. In the absence of bone injury the wound is sutured without drainage. If fracture is present, enlarge the wound if more room is necessary, a half inch trephine crown is raised, all loose fragments and any possibly infected bone are removed, using the craniectomy forceps if necessary. If the dura is uninjured the wound is closed without drainage, and mastic wound varnish and gauze applied. A firm compress of gauze is put in place for 24 hours. When the brain is lacerated, explore gently with the index finger, remove any fragments of bone or metal when readily accessible, place a drain into the brain and suture the wound as before.

Of the 118 cases closed by primary suture without drainage 114 healed by first intention, 3 showed slight superficial sepsis, and 1 broke down altogether.

The size of the wound at times offers difficulty in making proper approximation. This is overcome most generally by freeing the scalp for some distance around the wound by lifting the cranial aponeurosis with an elevator.

It is inadvisable to open the dura in the presence of a septic wound. In 30 cases in the Rouen area in which excision of the infected wound was first resorted to, the dura was opened without a death. In 3 cases in which the dura was opened without excising the area of infected tissue, cerebral abscess and death occurred.

The methods insisted upon are as follows:

- (1) Careful preliminary examination.
- (2) Early operation on every scalp wound however slight.
- (3) Complete excision of all infected tissues at the commencement of the operation.
- (4) Removal of all accessible foreign bodies from the brain.
- (5) Primary suture of wounds.

Gray, H. M. W.: *Observations on Gunshot Wounds of the Head.*
Brit. M. J., 1916, 1, 261.

The principles in the treatment of these wounds as deduced by Gray are as follows: (1) Infected gunshot wounds of the skull and brain require more careful consideration and prompt attention than similar wounds of any other part. (2) Sepsis can best be combated and prevented by early and complete operations. (3) Permanent disability can be prevented in most cases by the systematic removal of foreign material or displaced bone from the surface or substance of the brain whenever these are accessible to legitimate surgery. (4) By these precautions the immediate results in the saving of life and more rapid restoration of function, when possible, are better than those obtained by more conservative procedures.

The presence of any foreign body in the brain may not cause immediate disability, but sooner or later the brain is very apt to resent the presence of these bodies, and untoward symptoms develop. Fragments of bone, clothing, metal, etc., should therefore be removed as soon as possible after the receipt of the injury. The presence or absence of cerebral or cerebellar symptoms should not, in the average case, deter the operator from the radical treatment of these wounds.

In minor injuries the lacerated scalp should be excised and sutured. Primary union usually results.

In depressed fractures of the inner table contusion of the brain is almost certain to occur. *The dura should be opened in all such cases*, even when it is apparently normal, otherwise injuries to the brain substance may be overlooked and scar tissue form, which may cause future trouble. Furthermore, the injured brain substance, if allowed to remain untouched, may become infected and cause abscess, encephalitis, or meningitis. When wounds of the blood sinuses are present, it is thought advisable to remove depressed fragments of bone for two reasons: (1) Their retention may cause obstruction to the return of blood from some part of the brain, or (2) may lead to septic thrombosis.

As to drainage of the brain, as a general rule this should be avoided whenever possible. The presence of definite pus, infected blood clot, of inaccessible definitely infected foreign bodies, or profuse oozing would indicate drainage. Bacteriological examination of removed substances should be made; and if streptococci are found, the drainage should be maintained until these disappear from the discharges or become very few in number.

Several points are enumerated by the author: (1) There may be multiple injuries; therefore the whole scalp should be shaved. (2) The force causing the injury usually results in local injury; injury by contre-coup has rarely to be considered. (3) Fracture of the inner table almost always means injury to the brain substance. (4) A complete operation facilitates repair, gives better immediate results, and

tends to prevent troublesome sequelæ more surely than an incomplete one. (5) Death is due in practically all cases to the effect of sepsis on the damaged brain. (6) The aim in all operations should be to remove as much infected material and tissue as is feasible. (7) Foreign bodies act deleteriously in four ways: By direct effect on delicate brain substance, favoring sepsis, interfering with circulation, and causing scar formation. (8) It is highly important to prevent scar-tissue formation, whether on or in the brain. The nature of the injury, the amount of sepsis, the presence or absence of foreign bodies, and the treatment employed have much to do with the amount of scar formation.

The routine of treatment is as follows: On admission the patient's scalp is shaved, the wound thoroughly examined, and two skiagrams taken at right angles to each other, and an exhaustive neurological examination made. An aperient is given and urotropine given. If the brain is exposed, operation should be done at once, and in no case should operation be postponed longer than two days.

The majority of wounds of the scalp should be excised and the bone beneath carefully examined. If no bone injury is found, the wound can usually be sutured and primary union almost always follows.

Depressed fracture demands immediate exploration. Some cases without injury to the external table may have fracture of the internal table, usually suspected from the location of wounds or the clinical findings. Where the dura is normal in appearance and the brain pulsates well it may not be necessary to open the dura. When the dura is muddy looking and the brain does not pulsate it should be opened up by means of a crucial incision. The useless brain material will usually exude.

An injury to the dura without foreign body or sepsis requires careful trimming of the dura, the lost tissue being replaced by a piece of aponeurosis and the scalp sutured. Where a foreign body or sepsis accompanies the injury its withdrawal is attempted and drains usually inserted along the track.

Injury to the blood sinuses can often be closed by the application of a small piece of aponeurosis. The opening is carefully cleansed and the small piece of fascia then quickly applied.

Lumber puncture has given relief from persistent headache in many cases, but ordinarily no more than 20 cubic centimeters should be withdrawn.

The work of Don serves to explain away, in a measure, the seemingly divergent views of the radicals and conservatives. Don's work was done at a casualty clearing station (synonymous with the American Evacuation Hospital), and although he defends early operation, he is careful to limit the operative procedure to the barely essential

steps necessary to combat sepsis and compression. He specifically warns against the so-called early complete operation.

Don, A.: Treatment of Head Injuries in a Casualty Clearing Station. *Lancet*, Lond., 1916, cxc, 1034.

The author notes the treatment of 150 cases of head injuries operated upon since the war began, most of them in a casualty clearing station which was located in France not far from the trench fighting, and the cases came under treatment soon after the receipt of the injury. Casualty clearing stations are not fully equipped for work. They lack X-ray machines—a very important essential in the management of head cases. The results given by the author were obtained in the absence of X-ray evidence, and as good as they are, they would naturally have been far better with more complete equipment.

The experience in the present war upholds the rule of early operation in all head cases. Travel in motor ambulances is bad for head cases, especially in winter. Delay means extension of sepsis, and sepsis is responsible for the large majority of deaths, either immediate or remote. External scalp wounds, injuries to the cranium, dura, brain, and meninges all require early attention to prevent sepsis, and this can be given with better results, because earlier, where the casualty stations are than later on the line of communications.

The plan followed is to cleanse the scalp of all dirt, blood, and hair. The field of operation should be guarded by clean towels; the scalp and wound are next painted with tincture of iodine, and the wound is then excised freely, leaving a clean-cut edge which is undamaged to the eye. If there is obvious injury to the skull, trephining should be promptly done. A hole three-fourths of an inch in diameter is made at the side of the opening or fissure and the dura examined. The trephine opening may be enlarged with a rongeur, and if there is no blood clot, opening in the dura, or other injury nothing more is necessary. If the dura is injured, it is slit up and spicules of bone or blood clot are removed. Probing with a probe, catheter, or finger should be avoided unless definite evidence of the presence of spicules of bone, metal, or other foreign body is detected. It goes without saying that pressure from intracranial blood clot should be treated in the same way.

The flap incision, which was extensively used in the beginning of the war, and the removal of a big piece of the skull by the de Vilbiss forceps are not suitable methods to use at clearing stations, since they interfere with subsequent operations that may be deemed necessary.

Shell wounds are prone to be followed by brain abscess because of hair and dirt carried to the brain. The rifle-bullet wounds are less apt to be followed by abscess or other complications. Men with head wounds do not as a rule return

to the colors, and lodged bullets should be removed at home hospitals where brain specialists are to be found. It is different in cases where lodged shell fragments are suspected. They are much more apt to induce sepsis and its complications, so that when possible, even at a clearing station, the rule is to remove them.

An opportunity to study the results of English surgeons who believe in huge scalp flaps and large cranial openings, and those of the French surgeons who practice the linear or angle incision and small trephine openings, is interestingly commented upon in favor of the latter, which is considered far more appropriate in casualty clearing stations where lack of adequate equipment obtains.

Indications for operation are: (1) The presence of a penetrating wound of the head; (2) fitness of patient to stand a general anesthetic; (3) the presence of a surgeon with some experience in cranial surgery.

The average operator can always remove dirt from the wound by a clean-cut incision; open the cranium wide enough for the extraction of pieces of bone pressing on the dura or sticking in the brain, to favor drainage of blood or pent-up brain débris and to restore pulsation. These essentials involve but little shock; they require a minimum of time and they are attended with immediate results. When so treated, head injuries are followed by primary healing in most cases, and cerebral hernia is the exception.

Though gas infection is rare in head wounds, free drainage should be afforded by plenty of drainage tubes inserted wherever drainage is called for, even in the brain opening. Ample drainage precludes the possibility of dead tissue persisting in wounds, and when devitalized tissue is eliminated saprophites, like *bacillus aerogenes capsulatus*, can no longer thrive.

After battles only the mild cases should be transferred to the rear. The most serious cases should be retained for some time for treatment on the lines mentioned, which will put them in a position to bear the ill effects of transport.

Cushing, however, seriously interferes with the spirit of compromise noted in Don's work. Cushing condemns all routine treatment practiced in the first-line hospitals and advises a delay of even four or five days until the patient can be subject to a complete operation done under adequate auspices.

Cushing, H.: Concerning Operations for the Craniocerebral Wounds of Modern Warfare. *Mil. Surgeon*, 1916, xxxviii, 601.

Wounds of the head and extremities form a large majority of the total injuries in the present war, as shown by recent statistics. It has been clearly proved that specialization

in the treatment of wounds in this war is of the greatest value in returning wounded men to active service in a condition of comparative health.

The importance of all cranial wounds, however slight, is emphasized. Roberts found that in a series of 140 supposedly minor scalp wounds 41.5 per cent had skull fractures with more or less severe intracranial complications.

Cushing is strongly opposed to the routine treatment practiced at some first-line hospitals, by enlargement of the wound by a crucial incision, elevation of the depressed fragments, etc., and gauze drainage. He cites cases in which the results of this treatment have been unsatisfactory or worse. He believes that in cases of cranial wounds removal to the base hospital, where proper equipment, carefully planned operations, aided by the X ray, can be had, is the wisest course. He advises a flap incision away from the wound, thorough exploration, closure of the incision with buried galea sutures, supplemented by cutaneous ones (to be removed on the second day) to insure primary healing with scalp protection for the denuded dura or brain; if drainage is advisable, rubber tissue drains in the distant angles of the incision should be used, gauze never. Under this treatment the patient's chances are better, even after a delay of several days, than with an immediate operation at an ill-equipped first-line hospital.

The different types of cranial wounds from projectiles are described, with their characteristic symptoms. An important one is the median tangential or "gutter" wound, received on the vertex, involving the lateral expansions of the longitudinal sinus, causing stasis in the large cerebral veins. The symptoms are those of immediate bilateral spastic paraplegia in the severer cases—"longitudinal sinus syndrome." A mild case observed by the author showed weakness and spasticity of both legs. The milder cases, even with depressed fracture, may recover without operation. In the severer cases, with cortical injury, operation should be undertaken only under the most favorable circumstances, the operator being prepared to control hemorrhage from a bleeding sinus by implantation of raw muscle or vulcanized fibrin fibers; ligation to be avoided if possible. The same principles apply in the treatment of posterior wounds involving the occipital lobes and causing central blindness.

In general, the author believes that *good results follow a primary operation with closure, even four or five days after the injury; poor results with death from meningitis follow in cases treated at the front in the routine way and packed with gauze.*

The question of hernia cerebri and brain fungus has been one of the most perplexing problems to be met in the treatment of gunshot wounds of the head. Smith, Rawling, and Makins furnish excel-

lently full data covering this special field, both as regards pathology and treatment:

S. Smith, M. B.: Notes on the Treatment of Hernia Cerebri. *Brit. Med. Jour.*, July 22, 1916, p. 102.

Smith says that in 18 months' experience, with about 650 cases of gunshot injuries of the brain, one of the most perplexing problems was that of preventing or treating cerebral hernia. The significance of cerebral hernia lies in the facts that it warns us of increased and increasing intracranial pressure, and that unless a state of equilibrium be speedily established the patient will die from compression, causing paralysis of his vital centers. The progressive protrusion of brain matter through a small cranial wound may also produce further or new functional disturbance, not only because the herniated brain is disintegrated but because the fibers in its neighborhood are displaced, stretched, and often ruptured. It is not, for instance, rare to find hemiplegia supervene where a hernia develops, either in the frontal or post-parietal region.

A hernia, too, especially if large, acts as a direct menace by reason of the tendency for it to become gravely infected, and for this infection to spread to the underlying brain substance, leading to a generalized cerebritis, or to the spread of infection into the lateral ventricle. The danger of this latter event is all the greater because often there is a diverticulum of the ventricle in the hernia.

Serious infection of the hernia is most likely to occur in those cases where it is strangulated, as so often happens when the bony opening through which it protrudes is too small, the familiar "button" hernia, with its attenuated and constricted pedicle, being thus produced. Such strangulation of the protrusion is followed by softening and sloughing, and thus the facility for deep infection is increased.

It has seemed to me that the brain, perhaps because normally it is so effectually covered and protected by its bony envelope, evinces more than any other tissue of the body a tendency when exposed to become ulcerated and sloughy, and this should be given great weight when the question of operative measures comes to be considered.

It is of importance, apart from the danger of opening up the highly susceptible subarachnoid space, that the dural opening already produced by the wound should not be enlarged. Still more important is it not to incise the dura if it be found intact, as this procedure is accompanied by the grave risk of infecting the damaged, but not septic, brain underneath, thus leading to the very condition of affairs favorable to the formation of a hernia that we are doing our utmost to prevent.

As a means of diminishing the cerebral exposure, Smith found suture of the edges of the scalp wound after excision of the edges of the original wound only possible in a rela-

tively small proportion of our cases, the wound generally having been far too extensive and septic for the performance of such a desirable procedure. Moreover, where such a method of closure has seemed practicable, the tension necessarily produced would have made efficient drainage very difficult, and there is an obvious objection to a tight suture lying pressed up against the exposed brain, which we are trying both to protect and to drain.

It goes almost without saying that another eminently desirable factor in the prevention or limitation of hernia cerebri is the effective treatment of the ever-prevalent sepsis, both by the use of efficient drainage with frequent dressings where necessary, and also by the use of antiseptic lotions.

It is important to make use of gravity in the treatment of hernia cerebri and also in its prevention, and with this end in view we always, when possible, sit our patients up as soon as they are sufficiently recovered from the effects of the operation; this procedure is, unfortunately, impracticable in many cases. In those cases, however, in which it can be done, this maneuver has a remarkable effect in limiting the hernia, and will even cause it to subside altogether in many cases. It is specially efficacious in herniæ of the frontal region or near the vertex, where gravity has full play and the tendency for the brain is to fall away from the wound. It is also important that, by the free administration of morphine, heroin, or similar drug, the patient should be kept quiet and so prevented from bruising his hernia by knocking it against the head rail—a by no means rare occurrence. With this end in view, too, it is well to keep the hernia, if protruding to any extent, surrounded by a sufficiently large “buffer” of wool to prevent injury.

In spite of every care, however, hernia cerebri will always be met with in a certain number of cases, both following operation and also in those in which no operation has been performed. It is here that the systematic use of lumbar puncture is of the utmost value.

It is important, too, that the fluid be withdrawn slowly, almost drop by drop, as otherwise the hernia, with the too sudden relief of pressure, may sink back through the bony opening into the brain, leaving a deep cavity where before there was a large hernia. This is dangerous, for adhesions may be broken down and a septic meningitis lighted up. The intervals allowed to elapse, between which repetitions of the puncture are necessary, depend on many factors, some of which will now be discussed: If after the first puncture the hernia shows no sign of decreasing, and compression symptoms do not decrease, it has generally been our practice to repeat the tapping on the alternate days, until the pressure as shown by the manometer readings is brought down to within reasonable limits; in a few cases of high pressure with rapidly increasing hernia and progressive signs of compression we have repeated the operation daily, though this, in my experience, is rarely necessary, the tapping on

alternate days giving equally good results. If the fluid is clear, thus showing that there is, in all probability, no inflammatory process at work, it is permissible to wait longer before repeating than if it is turbid, crowded with leucocytes, and, maybe, giving growths of organisms on culture. One can not assume, however, that clear cerebrospinal fluid drawn off by lumbar puncture necessarily means absence of meningitis, for more than one of our patients have died in whom lumbar puncture gave clear fluid until the last, though subsequent autopsy revealed extensive suppurative meningitis.

Rawling, L. B.: Hernia Cerebri. Surgery of the Head, *Oxford Press*, 1915, p. 106.

There are two varieties of hernia cerebri, the aseptic form and the septic or ordinary type.

Aseptic hernia cerebri.—In consequence of the explosive effect of a bullet on the normal brain, if portions of the bones of a skull are carried away, then the rise of intracranial pressure will cause a certain amount of extrusion of brain substance. Owing to hemorrhage in the brain substance produced by the explosive effect, such a hernia tends to remain just as long as there is increased intracranial pressure (Horsley). It is argued that in a clean wound there may be such venous engorgement and cerebrospinal edema as will raise the general intracranial pressure to such an extent that brain substance, more or less normal, will protrude.

Septic hernia of the brain.—When the dura mater is opened in the normal individual, there is not the slightest tendency for the brain to protrude through the membranous opening. On the other hand, if the intracranial pressure be increased by tumor, hemorrhage, etc., the brain will at once protrude through the opening and will continue to do so until such an amount of brain matter has been extruded as will bring the intracranial pressure again to the normal condition. This is the result aimed at in ordinary decompressions, whether temporal, cerebellar, or in other situations. In such cases, however, the protruding brain is covered with some scalp constituents and the wound is clean. To such brain bulgings the term *hernial protrusion* is best applied. During such operations, also, if the opening in the bone and the dura be of considerable size, the brain will not bulge too suddenly, the pia and arachnoid membranes will not be ruptured, the brain pressure is fully compensated, and the pia and arachnoid being whole, there is but little tendency for the brain to become adherent to the scalp. In some cases, no doubt, the pia-arachnoid ruptures and surface vessels burst, but the bulging brain can still be covered with scalp. There is some risk of formation of adhesions, but the condition is still one of hernial protrusion, not hernia cerebri.

On the other hand, if the bone be extensively destroyed (or removed by operation), if the dura be widely lacerated (or freely opened), and if the brain be under considerable pressure by reason of contained fragments of bone, bullet, etc., especially when such foreign bodies are infective, then the injured brain will bulge outwardly, to an extent proportionate to the increased pressure, and will appear on the surface. This is the condition known as hernia of the brain (*cerebri* and *cerebelli*), the brain appearing on the surface as a red, granular mass, pulsating freely, slightly constricted at its base, and with some surface suppuration. The application of pressure leads to some diminution in size, but any more forcible attempts at such reduction in size results in headache, unconsciousness, and perhaps in the development of fits.

There is, however, a third condition to be described, *fungus of the brain*, an even more serious condition.

In such conditions the brain herniates through a small opening in the dura mater, the constriction at the neck of the hernia interfering with the venous return, both from the herniated mass and in the immediate neighborhood of the neck of the hernia. In consequence, both the hernia itself and the neighboring brain will be in a state of venous engorgement and cerebrospinal oedema, with consequent increase in pressure. The protrusion, therefore, will tend to continue to increase in size and more and more brain matter will become extruded. A vicious circle is established, the fungus increases—perhaps to such an extent that a great mass of brain substance may be extruded on to the surface, with terrible organic disturbances.

If, now, there is added to this condition the existence within the brain of fragments of bone or bullet, all infective, the tendency to herniation is all the greater. These facts suffice to explain the frequency with which we see hernia of the brain in this present war.

Fungus of the brain appears on the surface as a moist, cauliflowerlike mass, readily bleeding and freely discharging sero-purulent fluid. Pulsation is present, but not nearly so free and forcible as in hernia of the brain. The constriction at the neck of the mass acts in damping pulsation much in the same way as constriction at the neck of a hernial sac interferes with impulse on coughing. The application of pressure to the hernia has but little effect in the reduction of the mass. It should be added that the increase in size of the protrusion is always due in part to the development of granulation tissue, the growth of which is exceedingly free on the brain substance.

If, now, a section could be taken through the fungus, deep into the underlying cortex, it would be seen (1) that the narrow neck is more or less strangulated by the edges of the dural opening; (2) that the subjacent brain is softer than normal, discolored and congested, oedematous, and without any defined margins, shading off in the rest of the brain;

and (3) that the horn of the ventricular cavity of the brain, which is in closet relation to the protrusion, is expanded in that direction, and, in the more serious cases, that it actually passes without the limits of the skull so as to occupy the central portion of the hernia itself, even bursting at the apex of the protrusion, discharging a copious stream of cerebrospinal fluid.

Fortunately, both in hernia and fungus of the brain, meningeal infection is of lesser frequency than might be imagined. The "swollen" brain lies in close contact with the dura, obliterating the subdural space, and adhesions form with great rapidity between the other membranes, affording fairly efficient barrier to meningeal spread and infection.

Symptoms.—If the hernia involves the "silent" areas of the brain, frontal and temporo-sphenoidal lobes, there may be no special urgent symptoms, provided that the degree of protrusion compensates fully for the increased intracranial pressure. In fact, in many cases, the patient feels perfectly well.

If the protrusion includes the motor cortex, the results are disastrous—spastic paralysis of the contralateral side of the body, with apasia in the event of the left side of the brain being involved, with exaggerated reflexes, Babinski, and, later on, secondary contractures.

If the occipital cortex protrudes, the patient will suffer from hemianopia, etc.

In addition, as a general rule, hernia of the brain is associated with some mental symptoms—lethargy, headache, cerebral irritation, or actual insanity—varying in degree according to the size of the hernia and its physiological importance.

Treatment: Rawling outlines the following treatment for hernia cerebri:

Class 1.—Of hernia of the brain, purposely produced, fully compensative and not increasing.

Class 2.—Of hernia of the brain, resulting from injury or from operation, noncompensative and tending to increase.

Class 3.—Of fungus of the brain.

In *class 1* the desired result has been attained, the protrusion compensating for the previous increase of intracranial pressure. All that remains to be done is to keep the exposed brain clean and dry, favoring by rest its recession and closing over. It is to be hoped that the protrusion will diminish in size, perhaps actually receding entirely, as the degree of venous congestion and cerebrospinal edema lessens. In the event of the more favorable result, the skin will grow in from the sides, finally covering in the protrusion, after which some form of curved metal cap will have to be worn to protect the region from the effect of accidental blows, etc. If the overgrowth of skin is slow or imperfect, the surface may be skin grafted (after Thiersch's method). Recession may be aided by periodic lumbar puncture and, in the more intractable cases, by a contralateral decompression.

To keep the hernia "dry and clean" the region must be first cleansed with washings of hydrogen peroxide and fomentations, renewed every two hours. Fomentations of saline solution give the best results. When clean, fomentations are discarded and dry dressings applied, together with some antiseptic powder, e. g., boric powder. These dressings are renewed daily, the old dressing being washed off, the hernia dried, painted over with iodine, and fresh gauze applied. The brain itself is insensitive and the daily dressings are practically painless.

If the hernia is obstinate, refusing to decrease in size, it may be painted over every third day with a 40 per cent solution of formalin, the tissue necrosing and drying up. The destruction of the protruding mass does not make the condition of the patient any worse, so far as the functions of the region are concerned. These are already destroyed, and no further bad effects can develop.

In class 2 the hernia is increasing in size after apparent adequate and deliberate opening of both dura and brain. This occurrence results by reason of the infectivity of the brain or because, in addition, there are foreign bodies in the brain, keeping up the pressure and increasing the degree of infection. Thus a bullet in the brain, even though noninfective or of slow infectivity, produces such congestion and oedema as demands further protrusion, till fully compensated, though such desirable results are not easily attained. Again, a more highly infected bullet, or fragments of bone, may result in diffuse cerebritis or localized abscess, both of which conditions produce such "swelling" of the brain as results in an increasing protrusion. In such cases it is obvious that complete X-ray pictures will be of the greatest assistance both in determining the cause and indicating the line of treatment.

First of all, it is obvious that the exciting cause must be removed if possible, bone fragments or bullet being extracted and an abscess evacuated and drained. This being done, and a considerable protrusion remaining, the measures available are as mentioned in the previous section. The hernia may be painted with a 40 per cent solution of formalin or with alcohol every third day, and on the two intervening days with a 2 per cent solution of iodine in spirit. By these means the protrusion may necrose and shrivel.

The hernia may be shaved off, though usually growing again. Unfortunately, also, the protrusion often contains in its base the expanded and dilated horn of the lateral ventricle, and the shaving off of the protrusion will uncover that region to direct infection, in addition to permitting of the free escape of cerebrospinal fluid. Rawling has carried out this procedure on some few occasions, and has never seen any harm result, whilst in two or three instances the ultimate result obtained by the shaving process has been quite satisfactory. The mass is cut away flush with the sur-

face of the skull, bleeding being arrested by the immediate application of dressings and firm bandage. The wound is re-dressed daily. The protrusion may not redevelop, and healing may take place. In one instance he shaved away the hernia three times before it quieted down and scarred over. The protruding brain is quite useless so far as its functions are concerned. There is no objection to the shaving away of the herniæ from that point of view.

As a last resource, there is the question of conducting a contralateral decompression, with the object of relieving the intracranial pressure and recession of the hernia. The decompression should be conducted over the opposite temporo-sphenoidal lobe, for if that lobe does bulge outwardly there are no after ill effects. Thus in a right-sided hernia the operation should be a left subtemporal decompression.

In any case the decompression opening must be free, both as regards dura and bone. After the operation more pressure is applied to the hernia than previously. The "window" allows protrusion in another place and assists in the recession of the hernia, after which it may scar over or be grafted after Thiersch's method.

The ultimate result in all these cases will hinge largely on the cortical region involved. If possessing known function, that function will be permanently impaired or lost. Consequently, the more serious results are seen after herniæ of the Rolandic and occipital regions. With frontal or temporo-sphenoidal herniæ the patient may get quite well.

In class 3, fungus of the brain, the protrusion is gripped at its neck and continually tends to increase in size. Such being the case, the cause must be tackled. The condition is desperate and the following course must be pursued—the hernia shaved away, the margins of the dural opening defined, and that membrane freely slit up. There is, of course, the danger of infecting the meninges, but so far as Rawling's experience goes such a result is of unlikely occurrence with due precautions. After shaving the hernia away the whole region is sponged with iodine, and here and there a director insinuated for a short distance beneath the dura and the membrane slit up freely in several directions. The increased opening ought to relieve the strangulation. The future care and progress of the case is indicated in the preceding section.

Makins, Geo. H.: *Illustrations of War Surgery*. *Brit. Jour. Surg.*, No. 10, 1916, p. 263.

Makins cites two cases of fungus cerebri which improved under nonoperative treatment. Percy Sargent appends the following comment on this subject:

The term "hernia" is often used to describe this condition, but it is best kept to indicate the protrusion of uninjured brain through a large opening deliberately produced as a decompressive measure. The term "fungus" is con-

veniently retained to designate such protrusions of damaged semistrangulated septic brain and granulation tissue as are here illustrated.

The escape of brain matter from the cranial cavity, which is so frequently seen in the earliest stages, is due to the sudden increase of pressure caused by the blow, and to the traumatic cerebral oedema which immediately follows. If no additional factor were to come into play to sustain and increase this pathological intracranial tension, any initial protrusion would disappear with the subsidence of the traumatic oedema, and no fungus cerebri would form. Infection of the wound, however, results in the onset of a secondary inflammatory oedema, which keeps up and increases the general intracranial pressure. This leads to a further protrusion of the softened brain through the dural opening, which becomes more and more tightly plugged, and consequently the drainage of the damaged septic brain is increasingly interfered with. This interference causes a still further rise in the intracranial tension, so that a vicious circle is established. Unless relief is obtained either by natural subsidence of the inflammatory process, or by the establishment of free drainage, death ultimately ensues from meningitis.

A vertical meningitis spreading from the wound is not common unless the wound has been inadvisedly interfered with at a very early date, for the subarachnoid space rapidly becomes sealed off by adhesions which form between the dura and the pia-arachnoid, and this process is assisted by the fact that the swollen brain is pressed firmly against the dural opening. Within the first 24 to 48 hours, therefore, any manipulation of the damaged brain for the purpose of removing bone fragments, or any rapid lowering of the intracranial tension by lumbar puncture, is apt to be followed by a vertical meningitis spreading from the wound.

On the other hand, if, after this danger period is passed, free drainage of the damaged brain is not provided, the infective process extends deeply, the ventricles become involved, and a basal meningitis results.

Fungus cerebri therefore is an indication of the increased intracranial pressure which results from inflammatory oedema. When this elevation of pressure is progressive, the fungus increases in size, is tense, and pulsates but feebly; at the same time headache and optic neuritis are present, and these, together with other pressure symptoms, increase until death ensues. On the other hand, when the intracranial tension is relieved, the fungus becomes flaccid, pulsates freely, and shrinks in size more or less rapidly.

With an increasing fungus the treatment must be directed firstly toward controlling the general intracranial pressure, and secondly to draining the infected necrotic brain connected with the fungus; and the second object can be greatly assisted by accomplishing the first.

Local treatment.—Indriven fragments of bone should be removed, the utmost gentleness being used in order, firstly,

to avoid tearing adhesions and so opening up the subarachnoid space; and secondly, to avoid spreading infection more deeply into the brain substance and so toward the ventricle. If bone spicules have already been removed, it is well to explore the track again, as occasionally (but rarely) a small collection of pus may be discovered and drained.

Reduction of intracranial pressure.—This would naturally be best achieved by removing bone and opening the dura freely around the fungus; but such a course is forbidden on account of the practical certainty of infecting the subdural space. The other means available are (1) lumbar puncture and (2) contralateral decompression.

(1) *Lumbar puncture.*—This is the more easily applicable method, with the additional advantage that examination of the fluid removed gives information as to the presence or absence of generalized meningitis.

In these cases the cerebrospinal pressure is generally found to be considerably raised, and a large quantity of fluid can easily be removed. *It is well, however, not to allow more than from 4 to 6 drachms to escape at one sitting, lest the rapid disturbance of pressure within the skull should of itself lead to spread of infection.* The operation may, however, be repeated daily or every other day, and the intracranial pressure be kept under control by that means.

(2) *Contralateral decompression.*—When repeated lumbar puncture fails to control the fungus a subtemporal decompression may be done in the hope that the complete relief so afforded may allow the necessary drainage of the septic brain at the site of injury to take place.

Woodroffe confirms the results of the French school regarding the efficacy of cartilaginous grafts to close cranial defects. He is careful to specify the train of symptoms which call for grafting and to state that the symptoms are "often" cured. In civil life but little has been accomplished in this direction. Cushing limits grafting to a small and almost negligible group of cases. (Keen's Surgery, Vol. III, p. 251.)

Warren Woodroffe, H. L.: The Reparation of Cranial Defects by Means of Cartilaginous Grafts. *Brit. Jour. Surg.*, July, 1917, Vol. II, p. 42.

Cartilaginous grafts are recommended for those patients with cranial defects who show signs of "weakened cerebral defenses," and who present no contraindication to operation.

The symptoms most complained of are headache, vertigo, and sudden blurring of the vision. Though these are common symptoms of cranial and intracranial trouble, they may fairly be laid to the charge of the cranial defect when they are brought on by sudden movement. Inability to sleep, except with the head raised or tightly bandaged, and objec-

tion to noise, are also complained of. These symptoms can often be cured by a cranioplasty.

Technique of operation.—Make a crucial incision over the defect. Clean the circumference of the edge of the bone defect right down to the dura, and freshen the bone edges with a rongeur. It is rarely necessary to open the dura. A swab of hydrogen peroxide is placed in wound, and the flaps turned back over it.

The cartilages of the seventh, eighth, and ninth ribs having been exposed, shavings are taken of about half their thickness. Care should be taken not to cut through the entire thickness of a fixed cartilage; but the whole tip of a floating one may be taken. It is well to take what seems to be considerably more than enough cartilage to fill the gap. Each graft, as cut, is dropped into warm saline solution.

The chief difficulty is to keep these grafts from slipping. A very simple and rapid method is that of Villandre. One end of a fine catgut stitch is passed through the pericranium and tied. It is then passed through the pericranium on the other side of the gap and taken backward and forward in a zigzag manner till the hole is covered in with a trellis. In the case of a very large gap it is wise to supplement this network by a second, at right angles to it, and to insinuate the grafts between the two layers. We have now a small chamber bounded by the dura, the edges of the skull, and our trellis, into which the grafts can be slipped with a forceps. It is advised to apply the perichondral surface to the dura in order to avoid adhesions.

Careful wound closure, leaving a strand of silkworm gut in place for drainage.

Although the subject of shell shock belongs almost exclusively in the realm of neurology and psychiatry, we include an abstract of an article by Mott, which represents the best work on this subject. The original paper is very extensive and very detailed both from the clinical and the laboratory side. The surgeon must acquaint himself with the subject of shell shock for the reason that group 2 as outlined by Mott is distinctly a surgical group.

Mott, Fred. W.: *The Effects of High Explosives Upon the Central Nervous System.* *Lancet*, February 12, 1916.

High explosives contained in huge shells have played a prominent part in this war, and apart from the effects produced by direct material injury to the central nervous system, there is the moral effect of the continued anxious tension of what may happen, which, combined with the terror caused by the horrible sights of death and destruction around, tends to exhaust and eventually even shatter the strongest nervous system. To live in trenches or underground for days or

weeks, exposed continually to wet, cold, and often, owing to the shelling of the communication trenches, to hunger, combined with fearful tension and apprehension, may so lower the vital resistance of the strongest nervous system that a shell bursting near, and without causing any visible injury, is sufficient to lead to a sudden loss of consciousness. So that in considering the effects of high explosives it is absolutely necessary to take into account the state of the nervous system of the individual at the time of the "shock" caused by the explosive. A neuro-potentially sound soldier in this trench warfare may from the stress of prolonged active service acquire a neurasthenic condition, and it stands to reason that a soldier who has become neurasthenic from a head injury or from the acquirement of a disease prior to his enlistment will not stand the strain as well as a neuro-potentially sound man. Again, if in a soldier there is an inborn timorous or neurotic disposition or an inborn germinal or acquired neuropathic or psychopathic taint causing a *locus minoris resistentiæ* in the central nervous system, it necessarily follows that he will be less able to withstand the terrifying effects of shell fire and the stress of trench warfare. Thus, whether a tendency to a neurasthenic condition has been acquired or is more or less inborn, an emotional experience such as fright is more liable to develop the symptoms of a functional neurosis or psychosis.

The effects of high explosives upon the central nervous system fall into three groups:

(1) Immediately fatal either from pieces of shell, stones, rocks, or portions of buildings striking the individual, causing instant death, or the person may be buried from the explosion of a mine. Again, instant death must have occurred in groups of men from the effects of shell fire and yet no visible injury has been found to account for it.

(2) In group 2 we can place those cases in which the detonation of high explosives has caused wounds and injuries of the body, including the central nervous system, which have not been immediately fatal. The number of these cases which do not exhibit any of the functional disorders and disturbances characteristic of what is termed "shell shock" without visible injury, although such individuals have received most serious and fatal wounds from exploding shells, leads one to consider that in a large proportion of cases of shell shock without visible injury there are other factors at work in the production of the nervous symptoms besides the actual aerial forces generated by the explosive.

(3) The third group includes injuries of the central nervous system without visible injury; this group includes the functional neuroses and psychoses. As we know, one of the peculiarities of the functional neuroses is not only the sudden manner in which an emotional shock may engender a loss of function, but likewise the sudden manner in which it may be unexpectedly restored by a sudden stimulus of the most varied kind, provided there is an element of surprise. That is, attention is for a moment taken off its guard. The

causés of shock to the nervous system by high explosives may be considered under the headings of physical trauma—concussion or “commotio cerebri” by direct aerial compression or by the force of the aerial compression blowing the person into the air or against the side of the trench or dugout; or by blowing down the parapet or roof on to him, causing concussion; or a sandbag hitting him on the head or spine might easily cause concussion without producing any visible injury. Again, he might be buried and partly asphyxiated or suffer from deoxygenation of his blood by CO poisoning, for these high explosives generate considerable quantities of CO, which is inodorous and would not be recognized. A man lying unconscious or even conscious and partially buried and unable to move would be very liable to be poisoned by CO and not know anything about it; nor would the rescuers, as the poisonous effects of the gas depend upon the amount in the atmosphere and the length of time to which the individual is exposed to it.

The following abstract calls attention to the very necessary co-operation between ophthalmologist and neurological surgeons:

Greenwood, Allen: Fundus Examinations in a British Base Hospital.
London Ophthalmoscope, 1916.

A four months' experience during the heavy fighting of 1916 in making consultation examinations of the eyes of soldiers with head injuries for one of the largest groups of base hospitals of the B. E. F., an opportunity was afforded the writer of seeing a large number of interesting cases. In the majority of the severe head blows inflicted by glancing bullets, by shrapnel balls (the so-called furrow wounds), or by an impinging but not penetrating piece of shell casing, resulting in splintering of the inner table, or in brain destruction without any splintering whatever, increased intracranial pressure will sooner or later manifest itself, just as it will in the frankly penetrating and perforating wounds of the calvarium. One of the early signs of this increase intracranial pressure will be an optic neuritis which quickly increases to a typical choking of the disk. In these cases the neuritis from the start is of the choked disk or intracranial type, where the swelling is confined almost wholly to the nerve head even if it is raised several dioptries above the normal surrounding retina. This type of optic neuritis, which gives no clue to the side of the brain injured, may from its appearances be differentiated from the inflammatory type which is seen in cases that develop meningitis. In the inflammatory type besides the swollen nerve head there is an extension of inflammatory signs and œdema outward into the surrounding retina with hemorrhages and exudates giving the picture seen in the cerebrospinal meningitis of civilian practice. In this type the optic nerve showing the first signs of inflammation is that *on the side where*

the meningitis is beginning. Thus it is possible in some cases to differentiate between intracranial pressure and meningitis even in the early stages of these conditions. Where the nerve change is due to increased intracranial pressure trephining over the injured brain area, which allows for the removal of an extradural or intradural blood clot or disorganized brain substance or depressed bone, results in its rapid disappearance. A recrudescence of these nerve conditions would indicate a return of the intracranial pressure, demanding further interference. When, however, the inflammatory neuritis type is seen it usually indicates a purulent meningitis for which little can be done. For the injuries of the back of the head besides a fundus inspection there should be a careful testing of the visual fields. Such an examination will reveal many cases of varying types of hemianopsia from the complete homonymous hemianopsia to hemianopic scotomata and quadrant defects. Frequently it will be found in the long furrow wounds across the occipital region that the brain lesion as shown by the hemianopsia is opposite to the most severe portion of the scalp injury. For a treatise on the very careful working out of such fields as these the reader is referred to an excellent one by Holmes and Lister. (Proceedings Royal Society Medicine, June, 1916.) Some of the hemianopsias will recover following operative interference, while some will not. Where the hemianopsia is not accompanied by optic nerve changes and there are no other indications for operation, and the bone uninjured, trephining is not advised.

The writer at one time had three men in adjoining beds in his ward all with a left homonymous hemianopsia, due to injury in the right occipital region, and an accompanying choked where following a trephining marked improvement in the fields took place. Various paralyses of ocular nerves may result from basal fractures, and are interesting from the standpoint of localization and diagnosis.

The very latest fully expressed opinions regarding various phases of surgery in war may be found in two papers, published in June of this year (1917) by G. H. Makins and Bowlby & Wallace. Both of these papers specifically contrast the treatment of head wounds as practiced early in the war with the present-day treatment, and for that reason we furnish full quotations. Makins quotes freely from the experience of Col. Percy Sargent.

Makins, G. H.: Development of British Surgery in Hospitals on Lines of Communication in France. *Brit. Med. Jour.*, June 16, 1917, p. 800.

A great change has taken place since the commencement of the war both in the nature of the cases and in their actual number. This change depends on the one hand on the fact that a larger number of these injuries are retained and oper-

ated upon at the front lines, and on the other on the protection afforded to the head by the helmet. The early treatment of these injuries has already been dealt with; it suffices here to say that the patients which now arrive have either already been operated upon and are in good condition, or they come down already suffering from septic complications. The general lines governing the treatment of the latter class of cases have been admirably laid down in a paper in the *British Journal of Surgery* by Sargent and Holmes, and certain points in the technique of the operative procedure elaborated. These authors have also dealt with the anatomical and histological changes associated with traumatic injuries and infected wounds of the brain and their bearing on the surgical treatment of these conditions. Further examination of a considerable number of patients some months after their return to England proved much more satisfactory than had been generally expected. It was found that the proportion of patients who die after transference to England is small; later complications, such as cerebral abscess, are comparatively rare, and serious sequelæ, such as insanity and epilepsy, are much less common than had been foretold. In only 15 per cent of the patients examined, however, had more than one year elapsed from the date of the injury. It also appeared that many patients with foreign bodies deeply lodged in the brain recover, and are scarcely more liable to serious complications than men in whom the brain has been merely exposed and lacerated. These conclusions are obviously only tentative, but as far as they go appear hopeful.

Holmes and Sargent have also described a condition hitherto rarely seen and established a definite symptom syndrome for its recognition. It is characterized by an immediate spastic paralysis of the legs and frequently associated with spastic paresis of the proximal segments of the upper limbs; they have shown it to be due to occlusion of the superior longitudinal sinus or of the veins that enter it by a depressed fracture of the vertex of the skull. Experience showed the results of surgical interference with cases of this class to have been extremely unsatisfactory. Thus among 39 cases observed which were operated upon either by the authors or others 15 deaths occurred, while among 37 cases in which no operation was undertaken only 1 died before transference to England. While it is allowed that these figures have no absolute value, as naturally only the most serious cases were selected for operation, and in seven of the fatal cases direct injury to the brain was present in addition, yet the results emphasized the danger of operation. Moreover, the uncomplicated cases showed a remarkable tendency to improve, probably owing to the free venous anastomosis permitting a reestablishment of the circulation.

An important contribution to the localization of function in the brain has been published by Lister and Holmes, who from a study of a large number of cases with injury in the occipital region were able to determine the relative positions

in the cortical visual areas of the foci that subserve vision of separate portions of the visual fields. They bring forward strong evidence with regard to the site for the center for macular or direct central vision, of which very little had been previously known.

The following conclusions are come to:

(1) The upper half of each retina is represented in the dorsal and the lower in the ventral part of each visual area.

(2) The center for macular or central vision lies in the posterior extremities of the visual areas, probably on the margins and the lateral surfaces of the occipital poles.

(3) That portion of each upper quadrant of the retina in the immediate neighborhood of, and including the adjacent part of, the fovea centralis is represented in the upper and posterior part of the visual area in the hemisphere of the same side, and vice versa.

(4) The center for vision subserved by the periphery of the retinae is probably situated in the anterior end of the visual area, and the serial concentric zones of the retina from the macula to the periphery are probably represented in this order from behind forward in the visual area.

Holmes and Smith have recorded observations on the nature and localization of motor apraxia, or the inability to perform purposeful actions despite the preservation of movement and power and in disturbance of the faculty of localizing objects in the external world by vision.

Probably in no other branch of medicine have so many and such difficult problems arisen as in the treatment of wounds and diseases of the nervous system. Further, in this field an extraordinary opportunity has occurred to observe, analyze, and record the effects of local lesions, many of which are rarely, if ever, seen in civil life. When the results of this work are eventually correlated they must throw much light on the physiology and the symptoms of disturbance of different parts of the brain, spinal cord, and peripheral nerves, and thus increase our knowledge of the diagnosis and treatment of nervous diseases. Special arrangements have been made in order that cases under early observation in France should be sent to special hospitals in England, so that continuous records will be maintained of a very large number of patients.

Col. Percy Sargent furnished Makins with the following summary of his opinions:

The very large experience gained of gunshot wounds of the head has led to a considerable degree of modification in their treatment. Immediate routine operation, often incomplete and, in the absence of full neurological information and X-ray examination, sometimes unnecessary and even misdirected, is no longer widely practiced. It has long since been made abundantly clear that early evacuation of operated cases is often followed by disaster. As it is impossible to operate upon these cases and to retain them at the clearing stations for a period which renders transportation safe, more especially during times of great military ac-

tivity, the practice now generally adopted is to transfer them without operation as soon as possible to hospitals farther down the line. It has been made quite clear that surgical intervention is rarely required for the relief of cerebral symptoms, whether general or focal. Its chief aim is the prevention of intradural infection. On this conception all cases of gunshot wounds of the head fall into one of two categories, according to whether the dura mater has or has not been penetrated. *Nonpenetrating wounds* have a low rate of mortality, whether operated upon or not, provided that the surgeon respects the integrity of the dura mater.

It is customary therefore to do in these cases only as much as may seem advisable to insure speedy healing, such as excision of the edges of the wound, removal where necessary of bony fragments, and partial or complete closure of the gap in the scalp either by suture or by some form of plastic operation.

Penetrating wounds, on the other hand, afford more room for difference of opinion regarding their treatment. Individual cases continue to present difficulties even to those who have seen large numbers, but, broadly speaking, there is a consensus of opinion in favor of the following line of treatment: The wounds having been cleansed and dressed, the patient is transferred as soon as possible to a hospital where he can be retained for at least a fortnight after the operation. A complete neurological and radiographic examination is made and the operative treatment then directed according to the diagnosis thus arrived at. *In some cases of penetrating wounds no operation is indicated*, such as those in which a bullet has passed completely through the head, or those in which a bullet or a metallic fragment is embedded in the brain at a distance from a small clean entrance wound and is giving rise to no symptoms. Another class of cases for which operative interference is usually contraindicated is that in which the longitudinal sinus has been injured. Cases where a track from the scalp wound leads down to indriven bony fragments, or to an easily accessible missile, are operated upon, briefly, as follows: A moderately large flap is turned down after resection of all damaged tissue around the scalp wound; the bony opening is enlarged sufficiently to expose thoroughly the opening in the dura mater; the indriven fragments of bone and metal are removed under a constant stream of hot physiological saline solution; and the track is drained by a celluloid, metal, or rubber tube brought out through the original wound. In cases of more superficial cerebral laceration, where track drainage is unnecessary, the principle is employed of covering the denuded brain by some plastic operation on the scalp; in these circumstances drainage tubes emerging from the angles of the scalp flap are usually employed for a few days.

Opinions still vary regarding the advisability of operating for the removal of bullets or shell fragments. There is much evidence to show that these foreign bodies are well

retained, and, apart from the uncommon accident of late suppuration, cause no symptoms. It has been stated by more than one writer that bullets embedded in the brain move about under the influence of gravity. The evidence for this view is wholly unconvincing. Removal of bullets, even when the wounds have healed and the risk of septic infection thereby is largely minimized, must be, even in skilled hands, attended by an amount of damage which in most cases would have more serious neurological consequences than could the presence of an aseptic bullet.

Primary removal of a deeply seated missile carries with it the additional risk of septic infection. For these reasons the usual practice is to leave alone such missiles.

The treatment of indriven fragments of bone is more debatable. When driven into the brain by a missile which is itself retained, the bony fragments are rarely, if ever, more deeply placed than the projectile. When driven in by the impact of a missile which does not itself enter the cranial cavity, the bony fragments are rarely found so deeply situated but that they can be removed along the track with little, if any, additional damage being done.

With regard to the septicity of these indriven metallic and bony fragments, it has been found that a large proportion, when dropped into culture media immediately upon removal, fail to provoke any bacterial growth, either aerobically or anaerobically.

The question of the intracranial pressure has been the subject of repeated observation. Among the conclusions of practical importance which have been arrived at are the following:

(1) Apart from the rare instances of extensive intracranial hemorrhage, traumatic oedema, whilst playing an important part in symptomatology, does not reach a sufficient degree of intensity to endanger life.

(2) The instances of severe intracranial hemorrhage not rapidly fatal are very few; and even amongst these there is a certain number which surgical intervention is not likely to save. Experience has shown that an intracranial hemorrhage which is sufficiently severe to demand operative relief, and which can be recovered from, gives unmistakable signs of its progress. The operation can be deliberately planned and carried out with the definite object in view. Exploratory operations on the chance of discovering a hemorrhage are rarely if ever called for.

(3) *In case of intracranial pressure from secondary oedema which is causing severe headache and herniation of brain, this can almost always be controlled by lumbar puncture.* Occasionally contralateral decompression has been done for these cases and has afforded good results.

Such evidence as is at present available from the later results (six months to two years) is all in support of the general policy of treatment outlined above.

Bowlby, A., and Wallace, C.: The Development of British Surgery at the Front. *Brit. Jour. Surg.*, June 2, 1917, p. 719.

At the beginning of the war surgeons called upon to treat head injuries applied the ordinary rules of civil practice and operated on them at once. They were confirmed in their opinion that operation was right, since, apart from the mere physical defects, many patients seemed to be suffering from compression.

These operations were done both at casualty clearing stations and field ambulances, but the best method of operative treatment was as yet undeveloped, and the result was that many septic complications were seen at the base. Next, it was noticed at the base that cases which, from force of circumstances, arrived there unoperated upon, did better than those operated on at the front. This was attributed at first to faulty technique, and within limits this criticism was just, as the right operation was as yet undeveloped, both at the base and the front.

The observation was next made that if patients were kept quiet at the place where they were operated upon they did well, while cases operated on and apparently doing well were reported to have arrived in bad condition at the base when evacuated early.

It thus became obvious that there were two reasons for head cases doing badly: (1) The want of a good operation, (2) early evacuation of cases well operated on.

There were then two alternatives: The cases must be either operated on at the front and kept, or else evacuated as soon as possible to the base before operation; a patient must not be operated upon and evacuated forthwith. Two procedures were therefore adopted. In times of pressure head cases were cleaned up and sent to the base at once, provided they were fit to travel, and in quiet times they were operated on and kept at rest at a casualty clearing station for a week or ten days. Even this period of rest after operation proved too short, though the results were better than in earlier evacuation.

The next step was the establishment of special hospitals for head cases at the front. Advantage was taken of the fact that a head case before operation traveled well, and the special hospitals were placed in the back part of an army area. These hospitals were never subjected to the sudden pressure that may fall on an advanced casualty station, and consequently the cases could remain there for a long time. By this means patients experienced the advantages both of early operation and prolonged rest. The actual method of evacuation is as follows: The patients are brought from the trenches to the casualty clearing station as rapidly as possible. Here they are examined and dressed. If the pulse is slow they are sent on to the special hospital. If the pulse is rapid they are put to bed and evacuated later, should they improve. No special attention is paid to the type of wound—reliance is placed on the slow pulse as a sign that the patient will bear the journey.

The type of operation that has eventually been found most beneficial has been arrived at after many changes. Workers, comparatively far apart and not in direct communication, have evolved very much the same operation. At the front a small conservative operation was formerly practiced which experience has shown to have been a little too limited in scope. At the base there were two schools—one favored an extensive removal of bone and a scalp flap, the other an enlargement of the scalp wound and a limited removal of bone. Gradually the types of operations have approximated. It has been found that the removal of bone sufficient to expose half an inch square (1.27 cm.) of uninjured dura is best suited to most cases. Opinions still differ, perhaps, as to the comparative merits of making a flap or enlarging the scalp wound. On the whole, the flap is the best as a routine, unless the wound, as in the case of a horizontal one, is so situated as to compel the use of a very large one.

The recognition of the fact that *a slow pulse is not necessarily a symptom of compression* (for it may occur with a wide exposure of the brain), and that the symptoms, paralytic and otherwise, are not due to depression of fragments but to a destruction or commotion of the brain matter which is not remediable by operation, has also had an effect upon procedure. In the first place, a slow pulse is welcomed as a sign that recovery may follow, and it is not taken as a sign that operation is urgently needed, but rather that it is worth doing. *The recognition that depression of fragments is not the usual cause of the symptoms has also done away with the notion that their removal must be immediately undertaken.*

It is true that the sooner a dirty wound is cleaned up the better, but immediate operation is in many head cases followed by a great drop in blood pressure, so that *some delay may be actually beneficial* on this account, and Col. Sargent has pointed out that *for at least 24 hours after injury the brain is liable to be œdematous, and to extrude unduly if operated on while in this condition.* A moderate delay has also been said to do good in that it *allows adhesions to form between the dura and the pia mater*, thus lessening the chance of a spread of infection over the brain surface.

At the same time that the best type of operation as regards the scalp and bony defect was being evolved many other points were in the process of settlement.

1. Excision of the wound was soon decided on.
2. There was at first considerable discussion as to how far the brain should be explored for bone fragments on the one hand and the projectile on the other. Every one was agreed that an X-ray picture had become a necessity, and the opinion was gradually formed that a limited and intelligent search for bony fragments and other foreign bodies was beneficial, but that attempts to reach a missile which was deeply embedded in the brain was not justifiable. Results seen to have proved the correctness of this line of treatment, for frag-

ments of shell are reported to have caused little trouble provided their weight was not enough to cause pressure on the surrounding brain during movements of the patient.

3. The fact that many patients with head wounds suffered from septic complications, and the general demand for the drainage of all wounds, led at first to the employment of drainage in most cases of cranial surgery, not only of the scalp but of the brain also. The results of drainage of the brain were not satisfactory, and gradually it was abandoned, at any rate as a primary measure. The introduction of tubes was first omitted, and subsequently systematic attempts were made to cover in the exposed brain, the scalp being brought together over the defect in the bone and dura, either by simple suture, pericranial flaps, or relieving incisions formed by undercutting the scalp. A drain introduced under the scalp is still generally employed. This covering up of the brain seems to have been a decided success, and, although septic complications are still too often met with, they are less frequent than in former times. There has consequently been a great decrease in the number of cases of hernia cerebri.

4. There is still some difference of opinion as to whether small cranial depressions and linear fractures with slight inequality of surface, uncomplicated by symptoms, should be operated on in the first instance.

5. Most surgeons have accepted the recommendation of Sargent and Gordon Holmes that depressed fractures over the longitudinal sinuses should be left alone in the first instance.

6. Most operators are of the opinion that the dura mater should not be opened if found intact. The recognition that true compression of the brain is seldom seen has helped the formation of this opinion.

7. A general anesthetic may with advantage be replaced by the local use of novocain and adrenalin. If this method is adopted the patient is given either hyoscine and morphine or omnopon and scopolamine an hour before the operation.

Thus, by careful individual observation, and by the comparison of results, a method of treatment has been evolved which is applicable to all cranial wounds, and capable of modification in individual cases. It may be summarized as follows:

A primary cleansing of the wound. The transmission of the patient as soon as possible to the hospital where he will convalesce. The taking of X-ray pictures. The excision of the scalp and bone wound. A limited and careful removal of foreign bodies. The covering of the exposed brain. The closure of the wound, with superficial draining, and a prolonged rest in bed.

FRENCH SCHOOL.

The school of French surgeons are, on the whole, more inclined than the English to view head injuries in the light of demanding

immediate and radical operative treatment. It is easier to get a composite notion of French than of English war surgery, because at the last French Congress (*Bull. et Mém. de la Soc. de Chir. de Paris*, 1916-17) the various French surgeons recorded their views very clearly. By way of résumé, we present a digest of these views, made by Maj. Tarnowsky.

French School (*Symposium presented and resolutions passed by the Société de Chirurgie, Paris*).

All head injuries should be carried from the place where they fell to the place where they can be operated upon as rapidly as compatible with the military necessities of the moment.

All head injuries should be explored immediately upon arrival at the designated hospital, regardless: Of the hour of arrival, of the date and hour of the wound, of the statements on the diagnosis tag, of the clean appearance of the dressing, or of the patient's state of fatigue.

Head injuries should, whenever possible, be transported directly from the battle field to the evacuation hospital, because—

(a) Once operated upon, they should not be subjected to further transportation until they are convalescent;

(b) Because field ambulances and field hospitals are within range of artillery fire and the noise and concussion are very detrimental to such cases.

If primarily evacuated to a field ambulance or field hospital, the wound should be prepared surgically (shaving, scrubbing, trimming, and dressing) before further evacuation to the rear.

Radiography is of inestimable value, but a temporary breakdown of the apparatus should not deter the the surgeon from immediate exploration.

French surgeons strongly advocate Tr. iodi and ether in the surgical preparation of the case.

Head injuries should reach the operative table from two to six hours after being injured.

GENERAL TREATMENT OF CRANIAL INJURIES.

1. Whenever available the skiagraph should be used in order to determine the type of fracture and the presence or absence of missiles.

2. Thorough preparation of the surrounding area, carefully protecting the wound from further contamination.

3. Removal of all devitalized and lacerated soft tissues.

4. Removal of all small or sharp-pointed fragments of bone and preservation of large fragments.

5. If the dura is intact and no focal symptoms have developed, leave it alone and close the wound after replacing all large bone fragments.

6. If the dura is torn and the skiagram reveals the presence of spicules of bone or of one or more missiles, gentle

exploration of the lacerated brain tract with the gloved finger and removal of the foreign bodies can not further traumatize the tissues.

7. The giant magnet is often useful in the removal of metal fragments (except copper). Unless the exact angle of penetration of the missile is recognized and the magnet is so manipulated as to draw the missile out along this same angle, considerable additional trauma to brain tissue may ensue. The weight and bulk of the magnet also make it difficult to keep in the zone of the advance.

8. Severe hemorrhage or persistent oozing from one of the main branches of the middle meningeal hemorrhage will require double ligating with fine catgut. Persistent oozing from the pia or arachnoid is readily controlled by applying small pieces of muscle tissue.

9. Dural defects should be closed by pieces of fascia or dental rubber.

10. Intradural drainage should not be resorted to.

11. Wherever possible the wound should be sutured, leaving a small drain extending to the meninges.

12. The dressings should only be changed when they are saturated or the secretions have dried up, making the dressing uncomfortable.

INDICATIONS FOR TREPHINING.

1. No obvious signs of depressed fracture, but: (a) Entrance and exit wounds are far distant from one another. (b) Patient unconscious at time he received his injury. (c) Persistent headaches or giddiness. (d) Fracture of outer table without depression of same.

2. Depressed fractures without injury to the dura.

3. Fractures with injury to the dura.

4. Fractures with injury to the dura and presence of a foreign body.

Should bony fragments be replaced?

Small, sharp, irregular fragments should never be replaced, as they tend to shift about and traumatize the dura. Large depressed, fairly even fragments should have their sharp edges trimmed off and may then be sterilized by boiling (or immersion in ether) before being replaced.

If a trephine has not been used, punch a circular opening through the most dependent portion of the fragment for drainage. The skin flap will keep the bone in its proper position. The advantages of replacing such pieces of bone are:

1. Prevention of hernia cerebri.

2. Scaffolding over which bony or fibrous cells proliferate.

Should the intact dura be incised?

(a) Never if normal colored and pulsating normally.

(b) Invariably, if the surface of the dura is decidedly cloudy or blackened, tense, and nonpulsating, or it presents a circumscribed loss of elasticity (unequal tension). One

will invariably find a hematoma or a contused brain area or both in such cases.

Should a torn dural opening be enlarged?

Yes; invariably. Enlarge the cranial opening if necessary in order to expose normal dura. Make a semilunar incision through dura beyond the traumatized area; remove all lacerated dura. After completing the operation cover the dural defect by means of (a) fascia lata (best); (b) dental rubber.

Remember that a piece of fascia as large as the palm of the hand will contract down to one-half or even one-third of its normal size. It should either be tucked in under the bone or lightly anchored to the dura with very fine interrupted catgut sutures.

Dental rubber makes an excellent nonirritating protective membrane; it has also been used to cover peritoneal defects and contused arterial or venous walls (Matas).

What are the best methods for controlling intracranial hemorrhage?

(a) Pieces of muscle (autogenous) applied directly to the bleeding surface without making undue pressure.

(b) Coagulen Kocher-Fonio 5 per cent solution in sterile water, boiled not to exceed five minutes and freshly prepared.

Having enlarged the dural wound, what further steps are necessary?

(a) Removal of blood clots.

(b) Trimming of lacerated brain tissue.

(c) Removal of bone spiculæ, pieces of cloth, dirt, etc.

(d) Search for and removal of metallic foreign bodies which have been previously localized by the X-ray.

Immediate removal of foreign bodies is justified because:

(a) Track through brain tissue is already present.

(b) No further injury to brain tissue need be made.

(c) If wound is large, sepsis is already present.

(d) Abscess has not formed.

Secondary (late) removal of a foreign body presents the following disadvantages:

(a) Exploring through scar tissue.

(b) Additional laceration of brain tissue.

(c) Often have to operate in an infected area (abscess, etc.), with danger of dissemination of the infection.

(d) The presence of a metallic body within the cranial cavity is a constant menace to the patient.

TECHNIQUE OF REMOVAL.

(a) Use all proper means of localizing the foreign body by means of the X-ray. Of especial importance is an exact estimation of the depth of the object from the surface.

(b) Gently explore the sinus leading to the object with the index finger of left hand; locate the object with tip of finger.

(c) Pass a small spoon (gallstone scoop) alongside finger and engage foreign body in the scoop.

(d) Withdraw finger and scoop simultaneously, with the foreign body between the two. This minimizes traumatism of brain tissue.

(e) Should the metallic body be embedded in bone, gently push aside brain tissue by means of two or three groove directors, thus enlarging the sinus. Introduce straight or curved forceps and endeavor to loosen the object. If successful, withdraw forceps and proceed as in C. D. If not successful, a small chisel and hammer may be necessary in order to free the object.

Should intracerebral drainage be used?

No. It is dangerous and always irritating. Extradural wicks may sometimes be used. Drainage from skin to trephine opening is commonly used and maintained until all danger of sepsis is past. Drains should only be changed on definite clinical indications. Extradural wicks should be renewed every second or third day.

Late complications in head injuries usually caused by:

(a) Latest activation of an encysted abscess.

(b) Exuberant bony callus causing pressure symptoms.

(c) Meningeal adhesions or scar tissue within brain substance, producing circulatory disturbances.

The manifestations are innumerable and the treatment pertains entirely to the base hospitals. The French Army surgeons recommend that a trephined soldier should never be sent back to the firing line.

CRANIOPLASTY.

Cranioplasty is indicated in all large cranial defects whenever a tendency to herniation of type (b) manifests itself. As it is best to delay this operation in order to be certain that no nidus of infection lurks behind or that a spicule of bone has not been missed, this operation will only exceptionally be performed in the advanced hospitals.

The question of anesthesia has in a measure settled itself, owing to the fact that one large group of head injuries are followed by such deep unconsciousness that no anesthetic is necessary, and another group lends itself admirably to local anesthesia by virtue of a more or less profound obtunding of sensation consequent upon the injury. A third group requires one of the volatile general anesthetics, either as an adjuvant to local anesthesia, or used alone. Both the English and French use chloroform more commonly than do American surgeons, and judging from the literature, neither the English or the French have adopted intratracheal anesthesia, which has become so popular in America in operations upon the head.

This type of administration of ether lessens the duration of post anesthetic vomiting and is particularly convenient in that it removes the anesthetist from close proximity to the field of operation. The division of head surgery in the expeditionary American hospital will be equipped with intratracheal anesthesia apparatus.

The following abstracts of articles by Quénu, Couteaud and Bellot, and Charles and Charrier discuss the subject of anesthesia. A fair test of ethyl chloride in America may be said to have demonstrated that this anesthetic is far from being as safe as its advocates thought, and it will probably be used in the war zone rarely, and then with great caution. Chloroform has been in favor largely because of its rapidity of action and the difficulty of securing a sufficient supply of ether in the war zone.

Quénu, J.: Local Anesthesia at the Surgical Ambulance (*L'anesthésie Local en Chirurgie Crânienne*). Paris Médicale, Sept. 9, 1916, p. 229.

From a point of view of anesthesia, Quénu classifies his cases in three groups:

1. Those in whom coma is complete are operated without any anesthesia.
2. Those who are in a stage of excitement and can not be reasoned with are given either ether or chloroform.
3. The other cases are operated under local anesthesia.

It is not always easy to distinguish between group 2 and group 3, and it very frequently happens that the operation is started with local anesthesia and finished under general anesthesia. The extent of the cranial injury never serves as a deterrent to the use of local anesthesia, nor does the fact that the cranium is wounded in several places. The local anesthesia used as a rule is one-half per cent fresh solution of novocaine to which adrenalin is added in the proportion of 25 drops to 100 drachms.

The technique employed is the ordinary one of blocking off the operative area by a wide ring of infiltration. Aside from occasional vomiting during the operation not an unpleasant accident nor incident has been encountered.

Couteaud and Bellot: Injuries of the Skull by Projectiles (*Des traumatismes crâniens par projectiles de guerre*). *Bull. et mém. Soc. de chir. de Par.*, 1915, xli, 1110.

The authors give the histories of 29 cases of gunshot injuries of the skull operated upon by them. Sixteen of them were simple penetrating wounds, in 8 the bullet had passed entirely through the skull, and in 5 the bone had simply been pushed in on the brain, without perforation of the dura mater. In most of the penetrating wounds only fragments of bone were found in the brain; the bullets had not lodged in the brain. In such cases the bone fragments should be carefully removed and the wound drained, but there

should be no probing for foreign bodies. It is only rarely necessary to extract a bullet from the brain.

All of the authors' operations were performed under local anesthesia. They used a mixture of one part of 0.5 per cent cocaine and two parts of 0.5 per cent stovaine, with a few drops of adrenalin added. In addition to the avoidance of surgical shock and vomiting after the anesthetic, local anesthesia allows the patient to make certain movements and responses that are of assistance to the operator. Ten of the 29 patients died, a mortality of 34.5 per cent. Fifty per cent of the patients with bullets passing entirely through the brain died. All except one of the patients who died were in very bad condition when operated upon; they were either in pronounced coma or meningo-encephalitis had already begun. In the cases where there was loss of substance in the parietal lobes there was paralysis, but in the injuries of the frontal lobes there were scarcely any cerebral symptoms and the patients all regained a normal psychic condition.

Carles, J., and Charrier, A.: General Anesthesia with Ethyl Chloride in Military Surgery (*L'anesthésie générale au chlorure d'éthyle et la chirurgie de guerre*). *Prog. med.*, 1915, xlii, 748.

The authors find that ethyl chloride anesthesia is quite as valuable in operations of 45 minutes' duration as in those of 5 minutes, though it has ordinarily been used heretofore only in very short operations. It is particularly valuable in military surgery because of the saving of time. It only takes from a few seconds to two minutes for the patient to become anesthetized and about the same time for him to awake from the anesthetic. The toxic action is very slight; there is seldom vomiting, and, if any, it is much milder than after chloroform or ether; albuminuria seldom follows, and, if it does, it is slight in degree. This makes it particularly valuable in cases of shock, feeble pulse, etc. They have used this form of anesthesia in 200 of 700 cases operated upon during the past five months. In administering it several cubic centimeters should be given at first to obtain complete anesthesia; after that about 0.5 ccm. every three or four minutes. The three or four respirations of pure air when the mask is raised to give the ethyl chloride are generally sufficient to prevent asphyxia. There is no danger of heart failure, as with chloroform, and in the rare cases where there is difficulty in respiration a few movements of artificial respiration generally restore the patient.

Though analgesia is perfect the relaxation of the muscle is not so complete as with ether or chloroform, so that the latter anesthetics are still to be preferred for long and delicate abdominal operations, but in others ethyl chloride could be substituted with advantage.

The following four abstracts by Lapointe, Chavannaz, Latarjet, and Gayet all deal with the treatment of head injuries in the zone of advance. This is the field that furnishes so much debate regarding immediate operation versus transport back to better equipped stations. It is interesting to note that these four operators are unanimous in their advocacy of immediate operation, though none of them perform extensive or radical operations.

Lapointe, A.: Operative Treatment of Injuries of the Skull in an Ambulance at the Front (Le traitement opératoire des blessures du crâne dans une ambulance de l'avant). *J. de chir.*, 1915, xiii, 241.

Lapointe reports 127 cases of injury of the skull operated on in his ambulance. He practiced early and systematic operation in all cases, excluding only those that were so nearly dead that there was no hope. He made a crucial incision in the scalp wound, examined for fractures, removed any fragments of bone, irrigated with hydrogen peroxide, and dressed with iodoform gauze. Trephining was necessary only in comparatively few cases.

He divides the cases into three classes: (1) Those with superficial injuries, with or without injury of the dura mater; (2) those in which the projectile had passed entirely through the head; and (3) those in which the projectile had entered and lodged in the brain.

There were 47 cases of superficial injury without penetration of the dura mater; 7 of these died, one from a cause independent of the skull injury, leaving a mortality of 13 per cent. The mortality in the 48 cases with perforation of the dura was 56 per cent, or, eliminating the very bad cases, which would have died anyway, 51 per cent. Infection was the usual cause of death. Of the 7 cases in which the bullet passed entirely through the head, 6 died and the 1 who recovered was left with a paraplegia.

There were 25 cases in which the bullets had lodged in the brain. Operation in these cases was limited to extracting fragments and trying to limit infection; the projectiles were not removed; nevertheless the mortality was 56 per cent. Moreover, those who recovered are still subject to the danger of late infection from the projectiles. Lapointe thinks that as a result of the present war the idea of leaving such projectiles will probably be reversed, and it will be thought best to make immediate roentgen examination and remove them.

His experience shows the comparative harmlessness of extradural injuries and the terrible mortality of intradural ones. The mortality of all the intradural injuries together was 58.75 per cent. *Part of this high mortality was due to the fact that it was impossible to operate early enough*; only 22 of their 127 cases were operated on the day of the injury; the remainder was due to the insufficient first aid given. Scarcely any of the wounded men had been shaved around the wound before the first dressing was applied. The importance of this measure is shown by comparing the mor-

tality statistics of head injuries among the Russians, who had long hair, and the Japanese, who had their heads shaved. Better results can only be obtained by more efficient first aid and earlier operation.

Chavannaz, G.: Treatment of Fractures of the Skull at the Front
(*Sur le traitement des fractures du crane par armes à feu dans le service de l'avant*). *Bull. et mém. Soc. de chir. de Par.*, 1915, xli, 549.

Chavannaz gives brief histories of 59 cases of fracture of the skull operated upon by him; he has had 67 cases in all, but the others were too near death when received to be operated upon.

He advocates operation in all cases of fracture of the skull. If the fracture is large the edges are smoothed off with bone forceps; if the opening is not large enough for examination of the wound a trephine is done; the toilette of the wound is carefully made, and bone splinters are looked for, but sometimes they are overlooked because they have penetrated the brain tissue so deeply. Because of the danger of infection he touches the brain surface with a gauze compress slightly moistened with dilute tincture of iodine. Drainage was maintained for 48 hours with a rubber drain; gauze drains were used only when there were extensive lesions of the intracranial sinuses. Unless the patients were in complete coma chloroform anæsthesia was given.

Among the 59 cases there were 26 deaths and 33 recoveries—that is, 55.91 per cent of cases were successful. The patients were kept under observation three weeks or more. In 7 of the cases there were lesions of the intracranial venous sinuses, one of which was treated by ligation, the others by tamponing. Four of these 7 died. The accessory nasal sinuses were involved in 6 cases, and all of them recovered. Two of these patients also had injuries of the eye which necessitated enucleation. In 8 of the cases there was paralysis; 3 of these died, in 2 the paralysis disappeared, in 2 it improved markedly, and in 1 it persisted.

Latarjet, A.: Pathologic Anatomy of the Immediate Lesions in Penetrating Cranial Fractures Due to Projectiles (*Anatomie pathologique des lésions immédiates dans les fractures pénétrantes du crane, par projectiles de guerre*). *Lyon chir.*, 1916, xlii, 213.

The pathological studies which Latarjet made immediately after death in cases of extensive cranial fractures have led him to the conclusion that in the less extensive injuries which are susceptible of recovery it is necessary to intervene very amply and without restriction.

The details of several cases are given with photographic illustrations to show the mechanism and consequences of various types of penetrating fractures. The large experience gained from the study and results of treatment of such injuries has led Latarjet to adopt the following treatment in cases of severe penetrating cranial fractures.

1. Very wide trepanation, which is not limited by the extent of lesions of the dura mater, but by the extent of the destructive cerebral lesions, that is to say, which extends 2 or 3 mm. beyond the limit of lesions of the soft meninges.

2. Clearance of the cerebral injured area; removal of visible fragments; delicate cerebral exploration with light tamponade of the lesion by a tampon saturated with weak iodide tincture. This tampon is allowed to remain.

3. Insertion of a very fine meshwork saturated with iodide tincture between the endocranium and dura. When a fissure exists this mesh is extended as much as possible in order to establish a barrier between the dura and the fissured internal table.

4. Between the dura and the external cerebral face a similar mesh is insinuated. This meshwork excludes the cerebral area which is the center of it and fulfills a double purpose. It allows drainage of subarachnoidal hæmorrhage and is an obstacle to diffusion from the septic cerebral area into the subarachnoidal spaces largely open to contact as well as tending to the production of adhesences which also afford protection against the spread of infection. The meshes are resaturated with iodide by means of a tampon, and are allowed to remain in place until the fifth or sixth day when they are removed and replaced by others.

Since the adoption of this method of treatment there have been no deaths from primitive meningitis which previously had habitually caused the death of such patients in the course of the second week. Latarjet thinks, moreover, that the fear of a later reproduction of encephalitis is diminished by the isolation of the cerebral substance from the fracture and fissures. His procedure is the application to the brain of the general rule of war surgery: To clear widely, to convert the lesion into a surface wound, and to isolate the septic area.

He reiterates that clinical and anatomical results have convinced him that the extent of the trepanation must be governed by the extent of the cerebral lesion area and that an attempt must be made to exclude this area by isolating it from the osseous lesions and from its communication with the meningeal spaces and blood vessels.

Gayet, G.: Surgery of Penetrating Injuries of the Skull at the Front
(La chirurgie des plaies pénétrantes du crâne par projectiles de guerre dans les ambulances immobilisées de l'avant). *Lyon chir.*, 1915, xii, 618.

The absolute rule in the army corps in which Gayet has worked is to send all cases of head injury as quickly as possible to the surgical ambulances. Automobiles are sent directly to the dressing stations for them. *Whatever hour of the day or night they come in, they are immediately examined and operated upon.* It is generally agreed that the prognosis depends very greatly upon the promptness of the operation. No matter how slight the wound may be, it is

opened up. Two cases are described where there was apparently only a very slight scalp wound, but when it was opened up the bone was found to be cracked. If these patients had been allowed to go without operation, they would have died of meningitis.

Most of the author's operations were performed three to six hours after the injury. The objects of operation are to control hemorrhage and prevent infection. If the dura is found normal in appearance, it should be left intact; but if it is ecchymotic and does not pulsate normally, it should be opened and the brain examined. No probing should be done for deep projectiles, but a careful examination should be made for superficial ones, and they should be removed.

Hemorrhage from the sinuses is controlled by tamponing, from the meningeal arteries by ligation. Deep hemorrhage is sometimes difficult to control, but it should be done by ligation rather than by pressure if possible. Gayet does not favor the use of strong antiseptics on brain tissue, but iodoform gauze may be used. This should be placed only on the surface. Drains should not be inserted in brain tissue. The first dressing should be left on 48 hours. Patients with brain injuries should be moved as little as possible. They should not be transported for at least two months if it is possible to keep them that long. In the meantime they may be given treatment for any paralysis or aphasia resulting from their wounds.

Gayet has operated upon 198 cases, with 100 recoveries, 76 deaths, and 22 unknown results. The cases that he calls cured were under observation for several weeks, and when they were discharged the brain was completely covered in with epidermis or active granulations. He has heard from five of the worst cases after more than six months had elapsed and all are alive, though two are under treatment for slight aphasia or paralysis.

Témoin emphasizes the dangers of tangential wounds, and in so far he is in agreement with the conservative English school. He recommends trephining in every case of tangential wound accompanied by the minutest fissuring of the skull, owing to the frequency with which fissuring and fracturing of the inner table accompanies tangential wounds.

Témoin: Fractures of the Skull by Tangential Shots (Fractures du crâne par lésion tangentielle de la tête). *Bull. et mém. Soc. de chir. de Par.* 1915, xii, 1024.

Témoin calls attention to the frequency with which injuries of the scalp, apparently slight, are accompanied by fracture of the skull. After having had one or two sad experiences in losing patients with encephalitis when they had come in with apparently only slight scalp wounds he adopted the plan of opening up all scalp wounds freely and examining the skull. If there is the slightest fissure of the

external table he trephines at once. Any clots or fragments are removed and a small drain left in the wound. Among 33 patients with scalp wounds treated in this way, 29 were found to have fractures of the skull. All of the 23 who were trephined immediately after their arrival at the hospital recovered; of the 5 who were not operated upon until symptoms of brain disturbance developed, 4 died. Therefore he is an ardent advocate of immediate operation in skull injuries.

In the discussion Pauchet pointed out that in some cases where there is no true fracture but careful examination shows an ecchymosis of the external table of the skull, trephining will reveal the fact that there is a fracture of the internal table; therefore cases showing such ecchymoses or hemorrhagic spots should be operated upon. Tuffier agreed with him in this opinion.

On the subject of intracranial and intracerebral foreign bodies the French are inclined also to take a more radical view than do the English. The following three abstracts illustrate this point. The third abstract (Villaret and Beaulieu) shows the relationship between foreign bodies and the so-called late results of head injuries.

Abadie: The Extraction of Intracranial Projectiles in Two Stages: Trepanation for Access After Radiographic Location; Extraction Under the Radioscopic Screen (L'ablation des projectiles intracraniens en deux temps; trépanation d'accès après repérage radiographique; extraction sous l'écran radioscopique). *Bull. et mém. Soc. de chir. de Par.*, 1916, xlii, 3.

Abadie thinks that all intracranial projectiles should be removed as early as possible. Where the foreign body is very distant from the point of entry or anatomically placed in such a way that the point of entry can not be used as an approach, he uses the following technique. He makes a trepanation at a point selected on account of its proximity to the projectile and also to the vessels and other structures. The dura is opened sufficiently to permit the introduction of a forceps, and the wound is temporarily dressed. On the next day, or the day following, extraction of the projectile is carried out under the radioscopic screen, no anesthesia being necessary.

Abadie considers that his method of extraction, a day or two after the operatory incision, avoids many causes of possible infection, and reduces the maneuvers of extraction to a minimum.

Villandre, C.: Metallic Intracranial Foreign Bodies Apparently Tolerated (Corps étrangers métalliques intracraniens tolérés en apparence.) *J. de méd. et de chir. prat.*, 1917, lxxxviii, 129.

Villandre refers to a recent report of Marie in which he referred to 31 cases of wounded soldiers who not only retained a projectile in their brain with no irritation, but in

the majority of cases were not even aware of the presence of the foreign body.

Villandre does not agree with Marie's belief that it is unnecessary to seek a projectile when it is causing no trouble and that the presence of the projectile is less dangerous for its bearer than would be any operation for its extraction. *Villandre thinks that such tolerance of projectiles is apparent only, and that at any time grave complications may ensue*; that such projectiles still harbor microbic agents, the virulence of which may be great even after long months of apparent toleration; and that a well-conducted operation is not dangerous.

In the author's service 20 such cases with tolerated cranial projectiles were observed. The developments are shown in the following table:

Number of cases observed.....	20
Still in observation.....	3
In which a fistula was developed.....	3
Accompanied by abscess.....	3
With development of Jacksonian epilepsy.....	3
With hemianopsia.....	1
With tolerated projectile (3 aseptic).....	6
With projectile not extracted.....	1
Two of these cases (abscess cases) died.	

The author believes that metallic foreign bodies, whether superficial or deeply embedded in the interior of the encephalic mass, are a permanent source of danger and in the majority of cases should be extracted as rapidly as possible.

Villaret, M., and Faure-Beaulieu: The Grave Accidents of Late Appearance in Craniocerebral Wounds of War (Les accidents graves d'apparition tardive chez les blessés de guerre craniocérébraux). *Bull. et mém. Soc. méd. d. hôp. de Par.*, 1916, xxxii, 535.

The authors give particulars of 27 cases of cranial wounds out of a total of 256 which have presented grave accidents appearing several months after the traumas. These cases are classified under four headings: (1) Late epilepsy, (2) grave mental troubles, (3) meningeal infection and abscess of the brain, (4) late brain hernia.

Mental troubles and late cerebral hernia are exceptional. The most frequent and important results are late epilepsy and suppurative meningo-encephalitis. Meningitis and suppurative encephalitis were noted in four cases appearing from two to eight months after the traumas. The study of these cases has shown the important part played by the persistence of foreign bodies and metallic débris in the lesion. In the case of late epilepsy it is most interesting from the point of view of the lapse of time after the trauma. In one case this extended to 13 months, but most often it oscillates between 4 and 10 months.

Most of these cases have been trephined for the primitive injury.

The practical conclusions which the authors draw from their study of the cases are that in the case of men trephined or presenting traces of craniocerebral traumas, the future prognosis must be reserved even in the absence of flagrant symptoms of central nervous lesions. Systematic radiologic examination of the cranium should always be made to discover metallic débris or osseous particles; these are foci which provoke late grave results. Such men should not be returned to the front but kept at duty in the rear under medical supervision.

On the subject of hernia and fungus, Marchack, and also Leriche, emphasize the necessity of enlarging the outlet. Neither of these authors call attention to the danger of breaking down meningeal adhesions by such operative attacks.

Marchack: Cerebral Hernia (Hernies cérébrales). *Presse méd.*, 1916, p. 35.

Cerebral hernia may be distinguished as occurring either with or without subjacent abscess. In the first form the abscess must at once be opened so that it may not discharge into the ventricle. These patients almost all succumb to meningocephalitis. But the hernia without abscess is of more interest as it is susceptible of treatment.

Marchack thinks that the cause of these cerebral herniae is congestive oedema of the traumatized brain, and that the tumor projected across the insufficiently opened dura mater becomes strangled and adherent to the ring.

Such a hernia usually occurs within a few hours of injury. It is not due to hypertension, because its volume diminishes only very slightly after lumbar punctures.

Probably one-half of those with gunshot cranial wounds show hernia, especially if the injury is in the right parietal region and even if the osseous breach is small. In wounds of the frontal and occipital region hernia is rare.

In treating hernia Marchack has tried all methods from ablation by the thermocautery to simple compression. He thinks that the treatment of choice is after a certain period to enlarge the strangulating ring, remove bone fragments, etc., lavage with 20 per cent formol and compression. In the course of treatment patients show crises of Jacksonian epilepsy, but repeated lumbar punctures cause them to disappear.

Leriche, R.: Pathogenesis and Treatment of Precocious, Persistent, Cerebral Hernia (Pathogénie et traitement de la hernie cérébrale précoce persistante). *Lyon chir.*, 1916, xii, 448.

Cerebral hernia is a frequent complication of cranial wounds. A number of small hernias which follow trepanation disappear spontaneously without leaving any trace behind. Others, greater in volume, increase incessantly and death by progressive encephalitis is the customary termination. But frequently hernias are seen to persist without increasing or diminishing in size, remaining stationary

while the condition of the patient improves. If they are left untreated some may recover after a long lapse of time, but a much larger number die either from cerebral abscess or meningo-encephalitis.

This cerebral hernia is generally considered to be an indication of a deep infection and a fatal prognosis is accepted. Leriche, however, believes that this pessimistic view is the result of a false idea of the pathogenesis. In reality he says that these early persisting encephaloceles are frequently curable as they are the mechanical translation of a permanent local irritation resulting from an insufficient trepanation and therefore should be treated by an enlargement of the osseous breach until meningeal and cerebral healthy tissues are met with.

The hernia is a mechanical phenomena of tissue disorganization which is aggravated owing to the strangulation of the vessels by a very narrow bony ring; it signifies that the trepanation has not been wide enough, and that there is still a local irritating agent (fissure, superficial foreign body, etc.), or a large area of cerebral contusion which is not decompressed. Under these circumstances a new very wide trepanation is called for; this is the best method of reducing the hernia and curing the patient. The results obtained by such methods for true persisting hernias, in the case of patients otherwise in good state, are such as to suggest that early retrepanation is called for in patients who show an increasing hernia while the general condition declines. Nearly all such patients die from progressive encephalitis, and it seems to Leriche that here again the hernia is the result of an insufficient trepanation for the lesions, although often enough this trepanation is large. Moreover, by making a very large osseous breach, perhaps the number of deaths due to infection will also be diminished.

Leriche gives details and illustrations of many types of cases; considers the mechanism of recovery of hernias after wide trepanation; and gives the technical indications for trepanation in case of cerebral fungus.

The subject of grafting to fill in cranial defects has been exploited more extensively by the French than by any other surgeons. The following abstracts are fairly typical of many similar ones:

Morestin, H.: Cartilaginous Transplants in Reparative Surgery
(Les Transplantations Cartilagineuses dans la Chirurgie Reparatrice). *Bull. et. Mem. de la Soc. de Chir. de Paris*, 1915, xli, p. 1994.

Cartilages of the sixth, seventh, and eighth ribs are the only ones that can easily be made use of. Resection of any of these cartilages never brings about any untoward consequences if attention is given and if one reconstitutes with care by layers of sutures the musculo-aponeurotic bed. Even after having entirely removed the three cartilages mentioned, I have never observed any accident or incon-

venience whatever, and in particular neither lasting respiratory trouble nor difficulty on exertion, tendency to hernia, nor pain.

Costal cartilage easily lends itself to be cut with a knife. Sheets, rods, arcs, etc., can be cut and fashioned in the form for adaptation to their destination with extreme precision. The surgeon has no need in preparing his piece of prosthesis either of help or special instruments.

In spite of this property of adaptation, cartilage offers very great resistance and will take everywhere the place of bony tissue.

The transplanted fragments are really grafted, they live their normal life, they adapt themselves with extreme rapidity to their new conditions of existence. And this graft succeeds always, so to speak, whilst that of bone is so difficult to obtain. The grafted cartilage lives indefinitely without being resorbed, without undergoing appreciable diminution. The importance of this may be conceived. The result is that in practice one has not to consider reduction in size of the transplanted pieces, and one can and should give them their exact dimensions.

Cartilage may be taken from another person and may have been removed as long as a month previously to using as a graft. Operations can often be done under local anesthesia. The cartilage frequently remains slightly mobile on the bone, but is adherent to the soft parts.

After removing the costal cartilage it is necessary sometimes to ligate the internal mammary artery. After this the muscular bed, the superficial aponeurosis, and the skin are sutured.

In placing the cartilage the bony surfaces of the wound must be as free as possible of oozing of blood. The different pieces of cartilage should be applied to one another with flat surfaces. If bleeding persists, drainage must be provided for temporarily by leaving an opening in the skin wound, which is usually closed after 24 hours. As a rule, the cartilage is retained in place by being cut to fit accurately and needs no other retention.

Even infection of the operative field does not necessarily mean elimination of the cartilage, and it suffices to prevent its loss, to reopen the wound, evacuate the pus, and maintain drainage.

Leclerc, G., and Walch: Osseous Graft Taken From the Scapula to Replace Cranial Loss; Ivory Plates in the Repair of Cranial Losses (Perte de substance du crâne oblitérée par un greffon osseux emprunté à l'omoplate; deux observations de prothèse avec des plaques d'ivoire pour réparer des pertes de substance du crâne). *Bull. et mém. Soc. de chir. de par.*, 1916, xlii, 2021.

The above reports were submitted by Mauclair. In Leclerc's case the loss of cranial substance was repaired by a cranioplasty made at the expense of a bone graft borrowed from the scapula. The hole was the size of a 5-franc piece. In the cases reported by Walch the holes were approximately 5 cm. by 4 cm. and were repaired by ivory plates.

Mauclaire calls attention to the different methods of repairing cranial losses: periostic, osteoperiostic, and cutaneous periostic cranioplasty; (2) autoplasmic, homoplasmic, or heteroplasmic osseous grafts; (3) cartilaginous grafts; (4) transplants of macerated, decalcified, carbonized calcined or sterilized bone; (5) cranial prosthetics; (6) finally, in order to complete the repair, fat, serous, and fibrous complementary grafts.

All these methods have given good results, the cartilaginous is the most generally employed now, but time will show whether the cartilage becomes ossified.

Mauclaire reviews the history of the various procedures and thinks that generally speaking osseous and cartilaginous grafts are preferable to prosthetic procedures with ivory, metallic, or other plates. As regards functional results the end aimed at by the surgeon is not the amelioration of encephalic disturbance, but the protection of the brain from injury. The psychic effect is good because the patient feels that his brain is protected. The esthetic result is equally satisfactory.

Reports submitted by Marie, Claud, and Sicard do not, however, show that in cases of repair of osseous breeches that there is any satisfactory cerebral functional amelioration. Thus in 21 cases of repair on which Marie has reported there were 6 ameliorations without complete disappearance of subjective disturbance, 12 absolutely stationary, and 3 cases of aggravation of the subjective disturbances. Moreover, Mauclaire does not think it wise when there is a tendency to cerebral hernia to close the osseous breach. If there is hypertension of the cephalorachidian fluid it is best to defer repair.

The French have not studied the effects of shell shock and the consequent nervous phenomena as intensively as have the English, but the articles by Leriche and Baumel serve to emphasize the value of lumbar puncture for reducing the pressure of the cerebrospinal fluid in those cases presenting marked symptoms of injury to the central nervous system without demonstrable lesions.

Leriche, R.: Nonpenetrating Injuries of the Skull by the Bursting of Shells, and the Nervous Lesions Caused by Them. (*Des petites plaies du crâne par éclats d'obus et de bombes sans pénétration du projectile et des lésions nerveuses qui les accompagnent*). *Lyon chir.*, 1915, xii, 293.

Leriche gives an extensive study of the subject, reporting the details of 87 cases of skull injury and 11 others in which there was no direct wound but only severe concussion of the brain or spinal cord from explosion of shells or mines. He has seen 397 cases in which the fragment of shell had apparently only bruised the scalp. But no matter how slight the wound appears to be, an exploratory incision should be made in every case, and if the bone looks at all abnormal it should

be trephined; but if the dura is found intact it should never be incised, no matter how severe the subjacent hematoma and contusion of the brain may be. High pressure in the spinal fluid does not distinguish between deep-seated and superficial lesions. Leriche was inclined to believe that it did at first but has tested it thoroughly and found that it did not. High pressure and an abnormal tint of the fluid indicate the existence of small foci of contusion of the brain, due to air concussion from the explosion of the shell rather than to the effect of the projectile itself. *Lumbar puncture is of great value in treatment.*

In the cases caused by concussion the pressure of the spinal fluid remains high for days if puncture is not performed, and the patients show paralytic phenomena or melancholia with stupor or Jacksonian epilepsy. Edema of the brain and hemorrhagic foci were found on trephining, and lumbar puncture gave great relief.

Baumel, J.: Lumbar Puncture in Nervous Shock and Wounds of the Skull in War (La ponction lombaire dans les commotions nerveuses et les traumatismes du crâne par projectiles de guerre). *Lyon chir.*, 1915, xii, 271.

Baumel has had occasion to examine the cerebrospinal fluid in a large number of cases during the war, and he gives a table showing in detail the results in 56 cases. The condition of the spinal fluid gives important information as to the extent and severity of the injury. It is also of considerable value in treatment, for he finds that the pressure of the spinal fluid is above normal, even in cases of mere concussion where the projectiles have not come into direct contact with the skull. Nearly all the wounds reported were from fragments of shells.

If the spinal fluid shows an increase in the polynuclear count it means a more serious prognosis, for polynucleosis is an index of infection. When there is only lymphocytosis the meningitis is subacute. *Lumbar puncture is the only rational form of treatment in simple disturbance of the nervous system and in nonpenetrating wounds of the skull.* It is valuable also in cases of severe injury of the skull, as it reduces the symptoms caused by high pressure, rids the system of toxins, and hastens recovery. It should be performed systematically day after day as long as it is doing good, for it is absolutely harmless.

GERMAN SCHOOL.

A survey of the literature so far has shown that the English school is predominatingly conservative in setting their indications for operative interference, and also in limiting the extent of operative manipulations. The French are predominatingly radical along

these two lines. As will be seen in the following abstracts, the German school seems to be still in process of orienting itself. Articles by various men, following each other in close succession, furnish diametrically contrary advice. For example, the following abstract of Allers's paper seems to point most unequivocally against early operation in brain cases, and calls particular attention to the very important question of the influence of transport on patients with cranial injuries.

Allers: Transport of Wounded With Head Injuries. *Wien. klin. Wchnschr.*, 1916, Sept. 7.

Allers has studied the important question whether it is best to immediately operate upon cranial wounds at the advanced formations or to send them unoperated to the base hospitals. Sixty-six of these cases were operated upon at the front and then sent to the base.

Transportation within the first five days after operation means certain death; while the percentage of deaths falls gradually to nothing if transportation is delayed for four to five weeks.

Experience has shown that operation in cranial wounds deferred even up to three or four days after injury still gives good results. Allers finds that operations executed within the first week give a mortality of 12 per cent. It therefore seems best not to operate at the front; but if this is done then an interval of at least 10 to 12 days should elapse before the man is sent to the rear.

Allers thinks that the reason why the unoperated stand transportation better than the operated is to be found in the behavior of recent wounds. In such there is a very rapid formation of adhesions of the dura in the periphery of the injury. There is a concomitant increase of cerebral tissue as well as an increase of pressure, all of which tend to prevent diffusion of infection. But if fragments of bone, etc., are removed secretory flow and tension are diminished, and there is little obstacle to the spread of infection. Autopsy findings confirm this point of view.

The stand taken by Allers is supported by no less an authority than Tilmann, whose paper is valuable and meaty in that it discusses the value of mortality statistics, hemorrhage, inflammation, removal of foreign bodies, infectivity of the brain, and the performance of plastic operations. Likewise Friedrich supports the conservative stand, even going so far as to state that frank symptoms of compression do not demand immediately operation. Friedrich advises local anesthesia in all early operations.

Let it be very clearly understood that when these various operators argue against early operation their purpose is not always to

deny patients adequate, intelligent, and careful operative first aid, but merely to point out the necessity of doing as little work on the skull as possible, and in no way to tamper with the brain, before transport of the patient to a base hospital.

Tilman and Enderlen: Gunshot Wounds of the Skull (Schädel-schüsse). *Beitr. z. klin. Chir.*, 1915, xcvi, 454.

Tilman and Enderlen read papers on this subject before the meeting of military surgeons recently held in Brussels. They are in accord as to most points, though Tilman recommends at first only the necessary care of the wound, while Enderlen is an advocate of early operation. Percentages in regard to mortality are of no special value in these injuries, for many die later, after apparent recovery.

There is little danger of hemorrhage, for skull wounds bleed little. The greatest danger is that of infection, causing meningitis or encephalitis. There may be a nonseptic encephalitis from the inflammatory reaction of the brain to the presence of the foreign body, even though it is not infected. It therefore becomes a question whether there is greater danger in removing the projectile or leaving it. Operation should be performed only when aseptic treatment of the wound can be guaranteed.

The brain is very sensitive to infection and also to the action of disinfectants, so that their use in operations does more harm than good. Projectiles remaining in the brain should not be removed until their exact location has been determined by means of X-ray. In any necessary probing of the brain the finger should be used, rather than an instrument, for the finger can detect the difference in consistency between blood-clot and brain substance, while a sound can not. Operation on the brain, when necessary, can be performed without an anesthetic at all or under local anesthesia.

Meningitis should be treated by repeated lumbar puncture. Encephalitis is much more frequent than meningitis; the suppurative form is rapidly fatal. The serous, hemorrhagic, and reactive forms may recover. If the disease becomes chronic brain abscesses are formed, which have to be emptied by trephining. The nonsuppurative form of encephalitis may lead to softening and discharge of brain substance, or if the brain substance does not give way cysts may be formed; these may arise a long time after the injury. No patient who has had a brain injury should be transported for at least 8 days, even if there is apparent recovery. He should remain under medical surveillance for at least three weeks. Plastic operations are not advisable early, and even later they should be performed only when there are strict indications. Every effort should be made to secure healing by first intention, for it has been found that later epileptic attacks are much more frequent in cases where there has been a prolonged period of suppuration.

Friedrich, P. L.: Operative Indications in Gunshot Injuries of the Brain in War (Die operative Indikationsstellung bei den Hirnschüssen im Kriege). *Beitr. z. klin. Chir.*, 1914, xci, 271. (By Zentralbl. f. d. ges. Chir. u. i. Grenzgeb.)

There is a large percentage of gunshot injuries of the brain among the cases of death and of wounds handled during war. The methods in civil surgery, which are not uniform by any means, are not always applicable in war. Friedrich recommends that in injuries in civil life the wound be cared for at once, but only in exceptional cases should there be any operative procedure on the brain; the entrance wound should be left partly open, so that wound secretion, bits of necrotic brain, and foreign bodies may be discharged. An illustration is given of Thiersch's crown bandage, which leaves the wound free. From statistics of previous wars no general rules can be laid down as to war surgery, as the varying conditions must be taken into consideration. In war it is not a question of trephining but of operation on an already open skull. There are various kinds of injuries to the brain, and simple nomenclature should be agreed upon for the purpose of general understanding. As to depth, rebounding and grazing shots are distinguished; also open shots, either penetrating the whole skull, or making a unilateral wound; another classification is into wounds of the base or other regions of the skull.

In war a skilled surgeon should immediately look after the wound, but the skull should be spared as much as possible. Not all fragments need be removed, but only those lying free in the wounded area or those pressing against the brain. The degree of operation on the skull is illustrated by experiences in the hospital at Saloniki, where it was observed that too active operative procedures often produced bad results, while the results of expectant treatment were good. In closed injuries to the brain expectant treatment is still more indicated—at least attention to the entrance and exit wounds. Injuries to the base do not belong to primary surgery. Indications in rebounding shots, in hemorrhage inside the skull, and in the brain are discussed.

Even symptoms of brain pressure do not demand immediate operation if they are not progressive; sometimes even a technically correct early operation does not prevent late infection. The greatest reserve is also recommended when there are signs of cortical irritation in depressed fractures. Contractures are more of an indication for operation than convulsions. Disturbances of speech may appear even in injuries that are far away from the speech center. In all early operations general anesthesia should be abandoned in favor of local anesthesia in connection with morphine injections. When and where primary operations—that is, operations within the first 48 hours—shall be performed, depends on the means for transportation and care of the soldiers. Injuries of the skull and brain should be attended to as soon as possible either on the field or in its immediate neighborhood.

Among the late cases signs of brain pressure without infection are unusual. Infection predominates in these cases, and are to be judged by their clinical signs. A rise in temperature without any other cause serves as a warning. Operative interference should be undertaken through one of the wound openings. Several case histories are given.

Frey and Selye, Wilms, and Hosemann take a very radical stand, practically recommending early operation in all cases, stating their reasons and furnishing data on brain involvement, technique, drainage, foreign bodies, etc. Frey takes definite issue with those surgeons who claim that transport has any deleterious effects. Mueller's work is interesting chiefly from the point of view of statistics. He was stationed at a base hospital, and therefore not confronted with the problems of when and to what extent it was necessary to interfere.

Frey, H., and Selye, H.: Surgery of Gunshot Injuries of the Brain
(Beiträge zur Chirurgie der Schussverletzungen des Gehirns).
Wien. klin. Wchnschr., 1915, xxviii, 693, 723.

All cases of gunshot injury of the brain should be carried from the front to where they can get hospital treatment as quickly as possible, so that they may be operated upon at once. There is no particular danger of injury from the transportation. On the field a simple occlusion dressing is all that is necessary, and this should not be changed until the patient has arrived at the hospital. No definite conclusions as to the extent and depth of the injury can be drawn from the external appearance.

All wounds should be carefully incised and explored. If the bone is found intact, no further operation is necessary; but if the bone is injured the skull must be opened up. Enough bone must be removed so that sound and normal dura can be seen in all directions. After the removal of foreign bodies, splinters of bone, and crushed brain tissue, a cross-shaped incision is made in the dura, reaching to the edges of the bone. The wound must be dressed in such a way that the exposed parts of the brain are not pressed upon either by the dressings or by the natural coverings of the brain. Prolapse of the brain appearing later is of no significance if pulsation in it continues. If pulsation ceases the prolapse should be reduced and the brain explored again.

After serious brain operations the authors give urotropine, 2 to 3 gms. per day, internally, on account of its effect on the cerebrospinal fluid. When treated in this way the prognosis is very good. Only 8 per cent of the authors' cases died, but the time since operation is too short to report on permanent results.

Wilms: Treatment of Tangential Wounds of the Skull (Richtlinien in der Behandlung der Schaedeltangentialschuesse). *Muenchen. med. Wchnschr.*, 1915, lxii, 1437.

Tangential gunshot wounds of the skull demand early and thorough operation; fragments of bone should be removed and crushed, and softened parts of the brain carefully washed out with salt solution. Gauze strips wet with balsam of Peru should then be laid on the exposed brain to prevent infection. The treatment is very effective, as shown by the author's four years' experience with it. If operation is performed at once there is only the local injury of the brain to deal with; there is no increased intracranial pressure, but by the second day there is a diffuse oedema, which, even if it is not infected, tends to produce a prolapse. If operation is delayed this long a larger opening has to be made in order to provide for the discharge of the exudate, and it is more difficult to wash out the softened parts of the brain, for it is hard to distinguish them from the surrounding oedematous brain tissue. Of course the situation will be still more complicated if the exudate is inflammatory in nature. Wilms's work has been in a home hospital and he has had frequent occasion to see the bad late results of cases that were not promptly operated upon.

Puncture should not be performed for the sake of locating a brain abscess. New infection is introduced by the puncture needle as often as the abscess is located. Abscesses must be located by free incision, and this is especially true of the abscesses from tangential injuries, which generally lie very near the surface. In case of prolapse, which indicates increased intracranial pressure, extensive trephining must be performed. Wilms does not believe that gaps in the skull should be closed by plastic operation, at least not until a year or more after the injury, for the patient is for many months subject to the danger of late effects from the wound, and it should not be closed up by plastic operation.

Hosemann: Early Surgical Treatment of Gunshot Wounds of the Skull (Die chirurgische Frühbehandlung der Schädelchüsse). *Deutsche med. Wchnschr.*, 1915, xli, 607.

Hosemann had charge of a dressing station north of the Aisne for eight weeks. Injuries of the skull were extraordinarily frequent. He had 79 cases, and as there was time to give considerable care to each case they were treated at the dressing station rather than forwarding them to the hospitals. This is preferable if the conditions permit of it at all, for transportation is particularly dangerous in these cases. The hair was cut away, the wounds painted with tincture of iodine; and if necessary to get a clear view of the skull, the scalp wound was enlarged. In 24 cases this procedure showed that operation was necessary. Nine of these patients died.

There was very little infection among the cases—one case of meningitis and one of superficial brain abscess. *The brain is not so sensitive to infection as is commonly believed if it is given the necessary care early.* Another important point is to provide free drainage in order to avoid pressure on the brain. Dressings should be changed often so that the wound secretion may be discharged. Discharge of brain substance is not in itself especially dangerous; it is, however, an evidence of increased intracranial pressure, and indicates an examination for hematoma or brain abscess. The advice of some authors to close all defects in the skull by flaps of periosteum fascia, etc., is therefore based on a mistaken conception. It increases the very condition that is causing the brain prolapse. The indication in such cases is to keep the wound open, not to close it.

Mueller, F.: The Operative Treatment of Cranial Gunshot Injuries
(Zur operativen Behandlung der Schaedelschüsse). *Beitr. z. klin. Chir.*, 1916, c, *Kriegschir. Heft.*, 73.

In the last 11 months since Mueller took charge of the surgical division of the Tilsit Hospital, he has treated 180 gunshot injuries of the skull, among which were 11 through shots, 20 retention shots, and 146 ricochet and tangential shots.

There is no doubt as to the seriousness of the injury in segmental and diametrical gunshot injuries, where the brain is usually involved, but in tangential and retention gunshots it is only in a portion of the cases that serious symptoms are evident on inspection, and even the Roentgen examination is often not reliable. Such injuries may for a long time give no indication of dangerous injuries to the skull and brain. To await such symptoms leaves the patient in constant danger of his life, the attendants in continual worry, and the responsible surgeon cause to reproach himself later.

From these considerations Mueller decided to expose every cranial injury, even those appearing harmless. As a rule the situation becomes clear with one incision. He is fully convinced that a great part of his success is due to such primary intervention. The distribution of the 180 cranial injuries is shown in the table below:

Situation of cranial injury.	Total cases.	Deaths due to injury.	Mortality (per cent).	Recovered cases.	Recovery (per cent).
Soft-part gunshots.....	46	0	0	46	100
Extradural gunshots.....	22	0	0	22	100
Intradural gunshots.....	38	1	2.63	37	97.37
Brain gunshots.....	174	29	39.19	45	60.81

¹ Of these, 41 were primarily operated; 14 died, 66 per cent recovered; 33 were secondarily operated; 15 died.

Of the 180 skull injuries 132 were treated by early operation. Of these 60.5 per cent were operated upon within the first week of the injury, 29 per cent in the second week, and the remainder in the third week and later. Of the 180 cases 31 died, 30 from the direct results of the injury. Four patients died later in their home hospitals. Most of these deaths were caused by brain complications which were clinically of two distinct types. In the first the symptoms were of a fulminating character accompanied by high temperature and resulting in early death. The parts of the brain softened by the wound oozed continuously, indicating strong intracranial tension. It was not demonstrated whether or not there was a bacillar activity, but such assumption is plausible. In the second type the symptoms were insidious, the destructive process extending gradually until it reached a ventricle, when rupture occurred, followed by a suppurative ventricular inflammation and a bacillar meningitis.

From the percentages of mortality and recovery, the final success of the operative treatment can be established. First there is the noticeable result that of all extradural injuries there is an operative recovery of 97 per cent.

Of the gunshot injuries involving the brain 61 per cent recovered. Mueller's statistics show that after primary operation alone 66 per cent finally recovered. In those cases where a secondary operation was later necessitated there was only 33 per cent of ultimate recoveries. As against the 33 per cent recovered after secondary operation, the primary operated cases give a total recovery of 66 per cent. This comparison shows the importance of primary operation. Mueller thinks that in reference to gunshot skull injuries, success or nonsuccess depends upon the favorable issue of the first operative treatment. Retention gunshot wounds have a high mortality of 70 per cent, showing how destructive are the effects of a projectile remaining in the brain.

In primary operated tangential shots the mortality is 22.73 per cent, which gives the pleasing result that of 100 tangential shot injuries, 77 recovered owing to primary operation.

The article by Brandes is interesting from the fact that his data are based not only on the present conflict but also on the last Balkan war; and, furthermore, in that his stand furnishes a possible basis for interpreting the divergency of view of the radicals and conservatives. His dictum is: Base time and type of interference on type of missile.

Brandes: Treatment of Cranial Wounds. *Deutsche Med. Wchnschr.*, 1916, No. 23.

Brandes notes that the numerous publications on gunshot cranial wounds show a great diversity of opinion as regards treatment, especially as to wounds with arrested projectiles.

Many surgeons proceed only on the basis of their personal

observations, which are few. Some have abandoned conservative treatment and undertake operations varying from simple and superficial interventions to radical measures; others limit their operations to selected cases.

Brandes's experiences in the last Balkan War and in the present war have led him to proceed approximately according to the ideas of Holbeck and of Oettinger, i. e., *conservative treatment at first in wounds by arms of small caliber, and radical intervention in the case of shrapnel wounds, and to abandon this rule only in certain select cases.* His conclusions are summarized:

1. In the indications for operative intervention in gunshot wounds with projectile arrested in the brain (not in the cranium) we must clearly distinguish between projectiles of small caliber and those of artillery.

2. In case of brain lesions from small-caliber projectiles operation is performed only when there is evidence of beginning infection or progressive manifestations of cerebral compression, which call for intervention. Otherwise conservative treatment proceeds as advised.

(3) In shrapnel or grenade wounds with arrest of the projectile in the brain the author intervenes at once unless there is small probability of being able to immediately remove the projectile. He can not confirm either by his own observations or from autopsies. Holbeck's idea that in shrapnel injuries with the projectile arrested in the brain the projectiles exhaust their force in traversing the skull capping, since the bullet is often found at a depth of 2 to 3 cm. in the brain.

(4) Bier's method of causing the bullet to fall by blows against the head did not succeed in three cases in which the author tried it.

(5) Various theoretical considerations also militate against the probability of this method succeeding; besides it can not be considered harmless; it is less dangerous to intervene with the gloved finger to reach the bullet and then extract it.

6. If the bullet is not found at a reasonable depth in the brain, the author limits himself to tamponing the brain cavity and keeping the external aperture open in case of an initial encephalitis. Symptomatic prolapse invites intervention with good prospects; the encephalitis should be treated and the prolapsed pedicles freed by a wider removal of bone. The removal of the projectile can be obtained secondarily.

The work of Bárány, Jeger, Manasse, Marburg and Ranzi, and Bruns deals almost exclusively with the topic of infectivity of the brain and the development of traumatic brain abscess. Bárány's work is important as coming from a master in this particular field. What he says about closing brain wounds without drainage is therefore entitled to careful consideration, even though his views conflict with those of many of his German, French, and English colleagues.

Manasse recommends the gauze tampon, the use of which Cushing so unqualifiedly condemned in the treatment of brain injuries. Both Manasse and Marburg and Ranzi furnish details on the symptomatology of brain access. Bruns's article is included at this point, in spite of the fact that it deals largely with cord and nerves, for the reason that it also deals with the symptomatology and treatment of brain abscess.

The subject of brain abscess does not seem to be treated as adequately in the war literature as it is in the larger hand and text books of neurology and surgery.

Bárány, R.: Open and Closed Treatment of Gunshot Wounds of the Brain (Die offene und geschlossene Behandlung der Schussverletzungen des Gehirns). *Beitr. z. klin. Chir.*, 1915, xcvi, 397.

Bárány treated 60 cases of gunshot injury of the brain at a hospital in Przemyśl, where he had an opportunity to observe them throughout the course of the injury. In all cases he cut away the bone until 0.5 cm. of the dura was exposed, and then carefully removed fragments of bone and foreign bodies. In the first 39 cases he left the wounds open, draining with gutta-percha strips, which he found to be the best for the purpose. He had 8 recoveries and 31 deaths, or 20.5 per cent recoveries. However, 5 of the cases were in almost a dying condition when admitted, and subtracting these it gives 23.6 per cent recoveries.

After having seen some cases in which bullets had passed entirely through the brain and in which the patients had recovered without infection, it occurred to Bárány that bullet wounds of the brain might not necessarily be infected primarily, and if they are noninfected closed treatment is indicated to prevent secondary infection. In accordance with this idea he operated on 21 cases as before and sutured the wounds at once in 14 of the cases; the other 7 cases were complicated by injuries of the eye and nasal sinuses. Of the 14 cases 4 died, but they were in a hopeless condition when admitted. The other 10 recovered, and in 7 of these cases the wounds were so severe that he believes they would not have recovered under open treatment. He thinks the majority of cases that can be treated within 24 hours after the injury are not infected, and that the wounds should be sutured, thus preventing secondary infection. Of course if they are already so severely infected that abscess has developed they should be left open and drained.

Bárány: Primary Suture of Gunshot Wounds, Especially of the Brain (Primäre Wundnaht bei Schussverletzungen, Speziell des Gehirns). *Wien. klin. Wchnschr.*, 1915, xxviii, 525.

Bárány describes a number of cases of gunshot injury of the brain from which he draws the conclusion that it is better to suture at once without drainage. Theoretically

these wounds are to be regarded as infected, but practically they may be regarded as sterile and sutured. He believes, moreover, that in gunshot wounds in general much better results would be obtained if wounds were cleansed, the skin excised if necessary and sutured at once at the dressing station, than by the present method of simply dressing them and sending them on to the hospital. He thinks the wounded men would recover much sooner and be ready for military service again. Of course it would be necessary to simplify the procedure as much as possible. Instruments could be kept in alcohol all the time and the surgeon's hands sterilized with alcohol if water and soap were not obtainable. Excision of skin wounds could generally be accomplished under local anesthesia or without anesthesia at all. Practice would enable the surgeon to suture most wounds in a few minutes.

The objection is made that the patients would have to be transported and could not be under medical observation, but Bárány holds that they would not be any worse off than they are with their wounds simply bandaged. There would be even less danger of hemorrhage and infection, for the patient is exposed to both these dangers by displacement of the dressings during transportation. If the principle were once established that gunshot wounds should be sutured immediately, means could readily be found for carrying it out.

Jeger, E.: Plastic Closure of the Dura with Fascia in Gunshot Wounds of the Brain (Ueber primaere Fascienplastik bei Schussverletzungen der Dura). *Beitr. z. klin. Chir.*, 1915, xevii, 418.

Jeger was a coworker with Bárány in his work on gunshot injuries of the brain, and agrees fully with the latter's conclusions in regard to the superiority of closed treatment in such wounds. He thinks the good results may be due to the fact that the brain is placed under better physiological conditions when the wound is closed, and so is better able to resist any infection that may have taken place. As a further step in the closure of such wounds he replaced the defect in the dura with fascia in three cases, the details of which are given. The fascia prevents adhesions between the brain and skull, and the later results of such adhesions, as fistula and brain prolapse. Moreover, the physiological conditions are more completely restored when the brain is enveloped in all its coverings, and the fascia offers a still further protection against infection from outside. It also makes it possible to perform a secondary operation to repair the skull without exposing the brain. The fascia also has a hemostatic action that furthers recovery.

Jeger has thus far performed the above operation only in cases where there was such a large defect in the dura that there was danger of prolapse. The fascia is simply cut out and laid with its inner surface next the brain after the wound has been cleansed. The edges of the fascia are

pushed quite a distance in between the dura and bone. Sutures are not necessary. Considerable fat was left on the fascia and this helped to fill in the gap in the bone. There was healing by first intention in all the cases. He believes that primary suture in brain wounds with plastic closure of the dura brings about quicker and more favorable results than open treatment.

He also suggests as an operative possibility in inflammatory hydrocephalus externus a combination of a plastic operation on the dura, with the suturing of a piece of vein, one end under the fascia and the other in the external jugular, so as to drain off the fluid into the vein. He performed this operation in one case, but the patient was in too serious a condition to be saved.

Manasse, P.: Treatment of Brain Abscess (*Zur Therapie des Hirnabszesses*). *München. med. Wchnschr.*, 1915, lxi, 1475.

Much can be done to prevent brain abscess by careful treatment of all head wounds. They should be opened up thoroughly and examined and any foreign bodies removed. The wound should then be filled with loose gauze and left open. Skull wounds should never be sutured, as there are almost certain to be late complications if they are. Subdural hematomata should be treated conservatively; an intact dura should not be opened unless there are urgent symptoms. If a brain abscess forms in spite of these precautions it should be opened up freely and the pus drained out, care being taken to reach all pockets and recesses. Drainage should be provided with loose gauze; never with rubber or glass tubes. The first dressing should be left two or three days and after that the wound dressed daily. Each time the pus should be carefully but thoroughly sponged out. The patient must be placed in the best position for free drainage. He must be watched for any symptoms of retention of pus, such as fever, vomiting, headache, or localizing symptoms. If they develop the cavity must be palpated carefully with the finger to discover any recesses.

Sometimes secondary abscesses form in a prolapse; if so they must be opened up and if necessary the prolapsed part removed; if the prolapse shows no reaction it should be treated conservatively until it can be restored. If a fistula forms into the ventricle it generally results in basal meningitis and death. The abscess cavity granulates very slowly so that the patients have to remain under treatment for months, and they should still be kept under observation after complete healing, for they are very subject to later diseases of the brain. For this reason the author does not believe in plastic closure of gaps in the skull. He has treated 11 patients with brain abscess in a military hospital and 21 in Strassburg. Of the first group 5 recovered and 6 died, and of the second 5 recovered, 4 died, and 12 are

yet under treatment. In his five months' work in Strassburg Manasse had 265 cases of gunshot wounds of the head, in which 21 cases of brain abscess developed.

Marburg, O., and Ranzi, E.: Late Abscess After Gunshot Injury of the Brain (Über Spätabzesse nach Schussverletzungen des Gehirns). *Neurol. Zentralbl.*, 1915, xxxiv, 546.

The authors have operated upon 62 cases of gunshot injury of the brain during the past year, with 23 deaths. Among these 62 cases abscess was found at operation in 42. But there were a number of patients who apparently recovered perfectly from the operation, but who later developed abscesses and died. Six such cases are described. The abscesses generally developed four or five months after the operation; in one case the interval was eight months.

The symptomatology of late abscess is quite characteristic. The patient shows a rise of temperature for awhile, and then suddenly general symptoms develop, such as headache, vomiting, and signs of beginning meningitis. There is apt to be an increase in already existing local symptoms, such as hemiplegia or aphasia. These phenomena are explained by the fact that the abscess has been strictly encapsulated for some time, but finally there has been propagation of the pus to the meninges through a small opening into one of the ventricles. Often when the abscess becomes manifest it is too late to save the patient by operation, but cases can often be saved by early operation. An illustrative case is described.

From the foregoing it is evident that all cases of brain or skull wounds should be kept under careful observation for several months, and if there is a rise of temperature or the slightest sign of cerebral irritation the wound should be opened up. If there is pus, free incision and drainage are indicated.

Bruns, L.: Indications for Surgery in War Injuries of the Nervous System, and the Prognosis of These Injuries in Themselves and After Operation (Über die Indikationen zu den therapeutischen, speziell den chirurgischen Massnahmen bei den Kreisverletzungen des Nervensystems und über die Prognose dieser Verletzungen an sich und nach den verschiedenen Eingriffen). *Berl. klin. Wchnschr.*, 1915, lli, 989.

Bruns reports his experience at a base hospital with 376 cases of injury of the peripheral nerves, 89 of the brain and skull, and 37 of the spinal column and cord.

The injuries of the peripheral nerves he divides into three groups: (See Chap. III, Peripheral nerves.)

(1) In the first group the function of the whole cross section of the nerve is destroyed at the site of the lesion, so that all the muscles supplied by it are paralyzed; there is complete reaction of degeneration in the paralyzed muscles, and sensation in the region supplied by the nerve is more or less disturbed. It is impossible to tell whether the nerve is completely severed or whether it is only em-

bedded in scar tissue. The only way of finding out is to operate, opening up to the nerve and then proceeding according to the findings. Operation should be performed as soon as the wound is healed, any accompanying bone fractures consolidated, and all signs of sepsis disappeared. If during this period of waiting there has been marked improvement in the symptoms, operation may be deferred, in the hope of spontaneous restoration.

(2) In the second group of cases only some of the muscles supplied by the nerve are paralyzed, showing that the whole cross section of the nerve is not involved, but there is complete reaction of degeneration in the muscles that are affected. Operation may be deferred longer in these cases, for the lesion is less severe and they are more apt to recover spontaneously.

(3) In the third group the reaction of degeneration is only partial. These cases may be treated by electricity and massage. Neurolysis is especially indicated in those cases where there is severe and long-continued pain. Among the entire number of injuries of the peripheral nerves that Bruns has observed, there has been great improvement without operation in 33. He had great improvement after neurolysis in 13 cases, complete recovery in half of them. He has had successful results from nerve suture in 10 cases.

The lesions of the spinal cord (see Chapter II, Spinal Cord) are divided into those in which the whole cross section is injured, and those of partial injury. In the cases of partial injury operation should be performed only if the roentgen ray shows that fragments of bone or projectiles are compressing the cord, or if septic symptoms demand operation. In the majority of partial injuries operation is not indicated, and the prognosis without it is much better than might be expected. The cases of total injury are often so hopeless that operation is useless. There are cases, however, in which operation should be performed if the patient's condition permits it, though with or without operation the prognosis is extremely bad.

The skull wounds have mostly been treated before they reach the base hospital; that is, the wounds have been examined and cleansed and fragments of bone removed. These cases should be kept under observation for a long time at the base hospital, for every patient with a brain injury is in danger for a long period. If he develops signs of dizziness, headache, or nausea, his temperature should be taken and the eye-ground examined. If high tension of the pulse or mental dullness intervenes the wound should be opened up and an examination made for brain abscess. If bullets lodged in the brain are superficial they should be removed, for they always subject the patient to the danger of late abscess. If they are deep down they should be let alone, as the danger of exploring for them is too great; but it is often difficult, even with good roentgen pictures, to tell just how deep they are. The author has operated on 12 cases of brain

abscess with four deaths. After injuries of the cortex, attacks of cortical epilepsy are very frequent. He has not had sufficient experience to say whether operation is indicated for these. The prognosis with reference to mental defect after brain injuries is quite good. Recovery is seldom absolutely complete; for instance, after an aphasia there may remain slight disturbances in reading and writing; but if important association tracts are not involved in the injury the patients recover sufficiently to lead useful and active lives.

The work of Barth on meningitis was done before the war, but we have included it for the reason that it was Barth who stimulated a renewal of interest in the operative cure of meningitis. His 50 per cent recovery list is startling and has a direct bearing on the meningitis cases at the front. It must be added, however, that Barth's excellent results have not been duplicated in America.

Barth: Surgical Treatment of Suppurative Meningitis (Chirurgische Behandlung der eitrigen Meningitis). *Deutsche Gesellschaft. f. Chir.*, 1914. (By Zentralbl. f. d. ges. Chir. u. i. Grenzgeb.)

The author reports three cases of cerebrospinal meningitis which he cured by laminectomy of the lumbar vertebrae and drainage of the sac of the dura. The meningitis had developed after injuries. Staphylococci, diplococci, and streptococci were found in the fluid obtained on lumbar puncture. Before the operation puncture had been performed several times without results.

The prospects for operative cure in meningitis are not so bad as is commonly assumed if operation is performed early enough, for the disease begins as a diffuse process, and encapsulation of the pus between the cerebral convolutions does not take place until later. There are two reasons why there has been such great skepticism regarding the operation heretofore. Recovery was thought impossible, because only the terminal stages of the disease were being considered and because the course of such cases after operation was always thought of. It should not be forgotten that with the gradual development of meningitis, leucocytosis produces a stronger resistance to the infection. The spinal fluid obtained by lumbar puncture in meningitis has a markedly bactericidal effect, while this effect is completely lacking in normal cerebrospinal fluid.

The diagnosis depends on the presence of polynuclear leucocytes in the fluid from lumbar puncture; the infecting bacteria may have disappeared under the influence of the leucocytes. Recovery has been brought about surgically thus far in 50 cases, most of them in otology. Curability does not depend to any great extent on the bacteriological findings; cases showing pneumococci and streptococci have been cured.

Lumbar puncture should be performed on the very first appearance of symptoms of meningitis. There should be

immediate elimination of primary foci of suppuration, repeated lumbar puncture to relieve brain pressure, and if this is not sufficient, drainage of the cavity of the dura, either through the lumbar cord or the skull. Murphy drains in the posterior fossa above the foramen magnum through the cisterna cerebellaris. There is no rational ground for not treating meningitis operatively.

It has been practically impossible for the libraries of this country to secure any German medical literature of later date than early 1916. Through a private source the editors were fortunate enough to secure the 1916 and early 1917 numbers of the important German surgical journals. From these journals we have abstracted the following articles by Eiselsberg, Erdelyi, Axhausen, Peres, Guleke, and Joseph as representative of the latest and best German surgical thought. It is patent from these articles that, as war experience grows in Germany, the surgeons are veering somewhat more toward a conservative stand both in setting their indications for primary operative interference and in limiting the extent of their primary surgical procedures in treating gunshot injuries of the head immediately at the front.

Von Eiselsberg: Report on Gunshot Injuries of the Brain (Gehirn Schuesse insbesondere Spät Chirurgie). Report at the Second German Surgical Congress. *Beiträge zur Klin. Chir. Bd. V, Hft. 1. Kriegschirurgische hefte.* 1916. (Gehirn- und Nerven-chüsse, insbesondere Spätschirurgie.) Vorsitzender: General-arzt Prof. Dr. Enderlen. Erster Berichterstatter: K. u. K. Admiralstabsarzt Prof. Dr. Freih. von Eiselsberg. Zweiter Bericht-erstatte: Prof. Dr. M. Borchardt. Aussprache: die Herren E. Payr, Steintal, Fedor Krause, Kleist, Enderlen, Tilmann, Lud-loff, Lobenhoffer, Krüger, v. Gaza. Bruns' Kriegschirurgische Hefte der *Beiträge zur Klinischen chirurgie*. Zwanzigstes Heft. Fünfter Band. Erstes Heft. Verhandlungen der Zweiten Krieg-schirurgentagung, Berlin, April 26-27, 1916.

The treatment of skull and brain injuries belongs in large measure not to the surgeons in the first-aid stations, but to those at some distance. The majority of injuries of the brain are of such a nature that they prove immediately fatal. The others come under treatment several hours to a few days later. Primary operation is to be done particularly in fresh, tangential gunshot injuries *in which conditions permit*. Things necessary for proper treatment are (1) proper assistance, (2) proper armamentarium, and (3) a location such that the patient may remain on the spot and not be compromised by being transported for some days. When these conditions can be fulfilled, primary operation will secure much more rapid and smooth healing of the wounds, will diminish infection of wounds and diminish the danger of later infection, such as meningitis or encephalitis. Von Eiselsberg differs from Bárány, who recommends complete suture of the wound. Von Eiselsberg advocates universal drainage and loose suture.

When the skull injuries (and by skull injuries, Von Eiselsberg means, always, gunshot injuries) come to the base hospitals, the treatment will depend upon the symptoms which the patient shows. The chief danger lies in inflammation of the brain and its meninges. For the most part the patients will come under treatment at least 24 hours after injury and transportation. Under these circumstances, *unless symptoms demand immediate operation, von Eiselsberg believes it better to allow the patient a few hours' rest in bed, so that the general condition, the temperature, epileptiform seizures and the local conditions of the wound can be observed, and for X-ray examination in order to determine the extent of the bone injury.* Unless there are severe general symptoms or severe local symptoms, von Eiselsberg would not attempt operation without an X-ray picture. On the other hand, when there is profuse discharge from the wound or when the findings show a marked disturbance, such as a paralysis, then an immediate operation is to be recommended.

The technic of the operation is for the most part very simple. One can operate under local anesthesia, but ether anesthesia is to be preferred. The most important point is to ascertain the extent of brain injury. In order to do this, one should always bring as much of the injured brain as possible into the field of vision. One should remember, however, always to handle the brain as delicately as possible, and at the same time remember that it is much more dangerous to extensively explore the brain for splinters of bone than to allow such fragments to remain. It is a general principle that all foreign bodies *should be removed, because they can always produce abscesses.* Of all the instruments useful in exploration for bone fragments and foreign bodies, the very best is the little finger. With the smoothing up of the bone margins and the removal of foreign bodies, the operation is ended. The wound should be drained, *not packed*, a few stitches used, and a light, protective dressing applied.

When it is demonstrated during operation that there is no cortical pulsation, this is an indication of increased pressure, due either to hemorrhage or to inflammatory abscesses; and if hemorrhage is not present, then an exploratory incision should be made through the dura and into the brain. A good method of exploring the brain is by puncture with a thick needle; and the needle must be large because pus is often too thick to flow through a small needle. Perhaps still better is an incision with a pointed bistoury. When an abscess is located in the brain one should insert a small rubber drain for 24 to 48 hours. It should be remembered that pressure on the brain by pack or drainage tube will make a decubitus within a short time, and it is always necessary to remove drainage tubes within two days, or at least to shorten them.

When a brain abscess causes death, it is usually through progressive softening, or on account of perforation of the

abscess into the ventricle. Perforation toward with the meninges, with resulting meningitis, does not occur so often. When on exposing the brain, meningitis is found, then the opening should be enlarged in the hope that the meningitis may subside. Unfortunately, meningitis usually does not subside, or the improvement is only local. The purulent meningitis spreads toward the base and over the cortex and usually can not be influenced by operation. Multiple punctures of the brain do not seem to help. Spinal puncture serves mostly for a diagnostic and prognostic agent, rather than a therapeutic help. Urotropin does not seem to be of service after meningitis is well developed.

The prognosis is very different in abscess, provided circumstances permit the drainage of the abscess. Great difficulty is often encountered in localizing the abscess. Of 65 deaths that occurred under von Eiselberg's observation early in the war, 35 were the result of abscess. In these deaths from abscess death occurred for the most part on account of rupture of the abscess and sometimes on account of prolapse. von Eiselberg here mentions the observation of other men who believe that prolapse which does not show tendency to subside, speaks for an abscess or a severe inflammatory process in the depths of the brain.

In through-and-through gunshot injuries, von Eiselberg says, death usually occurs soon after injury, on account of the excessive brain destruction, and operation does not promise as much as in tangential injuries, because the entire path of the bullet is not accessible and removal of injured tissues is quite as impossible as in basal fractures. Even so, it may be very worth while to enlarge and clean the wounds of entrance and exit and remove whatever bone fragments are accessible.

A very difficult chapter is the one concerning projectiles which remain in the brain. These cases must be treated in the base hospitals unless the bullet lies directly under the skin. Naturally, very severe symptoms of pressure demand early operation, but in general it is much better that they be carefully X-rayed and the location of the projectile determined before operation. An extensive experience in peace time taught that in a large percentage of cases projectiles "heal in" without reaction, and it was the opinion that most projectiles were better unoperated. It has been the experience of each one, however, since the war began, that in the majority of cases the projectile causes a local abscess, which can be avoided by early removal. Occasionally the projectile is "healed in" without reaction, but the patient who carries a bullet in his brain carries, as it were, a "powder can," which can explode at any time. A very slight trauma, an infectious disease, or anything which will lower the patient's resistance will be an opportune time for a latent infection to develop a virulent character. Unfortunately, we have not yet learned in what per cent of cases the bullet will make later trouble, while in the cases with

acute symptoms the necessity for operation can not be discussed.

The surgeon often meets with the rather difficult question as to how far he endangers the patient by attempting to remove projectiles which cause no symptoms. *One may divide embedded projectiles into those which remain superficial and those which remain in the depths. Those which lie superficially can be easily removed and a practised surgeon may certainly attempt it. When they are in the depths, one always sees before his eyes the danger to life in operating.* Here the surgeon must determine for himself, after carefully localizing the projectile, whether it will be possible to extract the projectile without irreparably injuring the brain.

It is very easy to outline treatment of projectiles which are surrounded by abscess. The results of the operation, however, are much more doubtful. In 14 cases of foreign bodies with abscess, in not less than half the patients succumbed. This is quite sufficient to move one to search for the bullet at primary operation in order to avoid late abscess.

The patient with a projectile in the brain, even though without symptoms, should be kept quiet, and possibly through the rest infection will subside and the projectile will be better walled off. On the other hand, it should be repeated that even an encapsulated projectile, especially when it is one of the modern bullets or shrapnel bullets, is always a potential danger for the patient, and the patient should avoid every mental and physical exertion. As X-ray technic is improved and the bullets can be accurately localized, much good can be done and the mortality very distinctly reduced through early removal of projectiles without destructive operations.

von Eiselsberg again states that, in his opinion, the most difficult chapter in the treatment of gunshots of the skull is that concerning the inflammatory changes of the brain with the complications of abscess and meningitis.

The after treatment is also important. There can be no restoration of the brain tissue itself, and against the progressive inflammatory softening one is absolutely helpless, even as he is against the meningitis. These two factors are responsible for most of the bad results.

Another cause of bad result is prolapse, against which the surgeon is also almost helpless. The prolapse can be caused by a deep-lying abscess, and through drainage the condition may be improved. Prolapse may, however, be due to a progressive inflammation of the brain, coupled with a marked edema, and in many cases this progresses until death takes place. From a prognostic standpoint, one may divide prolapse into two forms, the unfavorable, which resist all treatment, and those which come spontaneously to healing or heal after opening an abscess.

Concerning the treatment of prolapse, there is no unity. Obviously, a pus focus should be drained. If necessary, the

opening in the skull should be enlarged. The dressing should be so applied that all pressure on the brain is avoided, and no attempt should be made to hold the prolapse back by the use of plates of bone or metal. The prolapsed portion should not be removed. Best of all is simply to allow the prolapse to remain undisturbed, and as the inflammatory processes subside the prolapse will disappear. A light loose dressing and daily application of tincture of iodine is all the treatment necessary.

The later complications, such as paralysis, disturbance of speech, etc., should be treated by medico-mechanical movements, speech lessons, etc.

Of special importance is the question of covering the defect, and one asks: "In which cases shall one repair the defect in the skull and in which ones not?" These questions are more important than the question of how shall one cover the defect. *While it is not necessary to repair small defects, it is certainly better when the brain is again normally inclosed in its hard capsule.* Headaches, so long as they lead one to suspect the possibility of abscess, are a direct contra-indication to plastic operations; also a purulent discharge from the wound contraindicates operation. The scar is often sufficient cause for repair, if it is bulging or painful or shows a tendency toward easy injury. A tendency toward epilepsy is a sufficient indication. The cause of the headaches is not in all cases an increase in brain pressure. It has been shown that in most cases there is an increase in pressure of cerebrospinal fluid; in some cases it is diminished.

There are two schools in regard to the point of doing skull plastics for epilepsy. In one, Kocher warns against covering a defect in the skull when epilepsy is present, and he relieves pressure by puncturing the ventricle; while Bunge seeks to restore the normal conditions of the skull and to cure the epilepsy by repairing the defect. Von Eiselberg is of the opinion that a plastic repair of a defect in the skull will often bring more danger than allowing the defect to remain open. One should always attempt, by giving bromide preparations, salt-free diet, and continued use of luminal, to control the epilepsy.

The operation should have as the objective, before all things, to prevent scar formation and adhesions between the brain and its coverings. Through the implantation of celluloid, or of fat, or of omentum, one should attempt to avoid the reformation of the scar.

von Eiselberg reports 27 cases of operation for repair of skull defects, of which 25 healed after operation, one required reopening of the wound, from which the bony flap was removed, and one died of later abscess. Twenty of these 27 cases were repaired by means of a free transplant of bone with periosteum from the tibia. The plastic operations were undertaken at varying periods after the injury, the majority at the sixth month. *One should wait a long time before closing the bone defect, on account of the*

danger of development of late abscesses. Of 16 cases of late abscess, von Eiselberg mentions three cases in particular, who were apparently well at six, eight, and ten months, respectively, after their injury, and then developed abscesses which led to death in spite of the fact that they were properly diagnosed and operations were made.

von Eiselberg did not see any bad results from taking the transplants from the tibia. Lately he has used some subcutaneous fat between the transplant and the brain, or he has put the periosteal surface toward the brain. Of 27 cases in which the transplanted bone healed in place, 24 healed fast, and in one the transplanted bone is still movable.

von Eiselberg quotes from Lexer, who found pathogenic bacteria in scars many months after operation, and on this account he would not attempt secondary skull plastics under one-half year, or, better, one or two years.

In von Eiselberg's opinion it can not be sufficiently emphasized that patients with skull injuries must be under careful medical observation for a long time and, when possible, adjacent to a surgical pavilion, and every exertion must be avoided because the danger of late abscess hangs always over their heads. The vast majority of these patients must be discharged as unfit for duty. He offers the following conclusions:

1. The most important and most dangerous complications of gunshot of the skull are, after the primary disturbances, the inflammatory processes of the brain and its meninges. Brain abscesses are always to be operated. In brain softening and meningitis, an operative attack is almost hopeless, and the same holds true for prolapse, except those caused by abscess.

2. All tangential gunshots, which show general clinical symptoms or local symptoms, or show no tendency toward improvement, are, especially if the X ray shows positive findings, to be operated.

3. Through and through gunshots in certain cases are better not operated. If operated, the attack should only be made in the attempt to prevent progressive inflammation and infection.

4. Projectiles lying superficially should be removed. The deep-lying one should be operated if the patient develops symptoms. The X ray is an invaluable help. When the projectiles heal in the depths without symptoms, then one must determine according to their location whether or not operation is to be done.

5. Epilepsy which occurs in connection with a defect in the skull should first be handled by inner medicaments, and only when this is without result and at a later time can they be operated. At later operation, through interposition of fat or celluloid plates, one can attempt to cure the epilepsy.

6. With the attempts at repair of skull defects, one should wait at least half a year after the subsidence of inflammatory symptoms.

7. All patients with gunshots of the skull should be under careful observation for a long time after complete wound healing, and whenever possible they should not be discharged, but kept in military hospitals, in order that they may be protected should the development of late abscesses and epilepsy occur.

8. Patients with gunshot injuries of the skull, in which the brain has been injured, should almost without exception be eventually discharged, unfit for service.

Erdelyi, E.: Gunshots of the Head (Über Schädelsschüsse). Kriegschirurgische Hefte *Beiträge zur Klinischen Chirurgie*. *Vierter Kriegschirurgischer Band*. (Kriegschirurgisches Heft XV.) May, 1916. P. 57.

Gunshots of the skull are in the majority of cases so severe that they lead to early death, and the patients suffering them remain upon the battlefield. Of those that live and submit to treatment every one should be regarded as infected and every means must be employed to prevent the wider spread of the infection.

Small, seemingly harmless wounds, with sharp entrance and exit openings, may be accompanied by the severest brain and nerve injuries. Therefore, every gunshot of the skull is a case for operation, even if the operation is nothing more or less than a thorough toilet of the wounds.

Exposure of the area of injury, removal of foreign bodies and bone fragments, and establishment of free drainage are recognized as good practice in both military and peace surgery. Furthermore, operation should be done as rapidly, as thoroughly, and as ideally as possible. Work half done is worse than absolutely none, and a late operation, undertaken after a prior operation poorly done, renders the prognosis extremely bad. For the most part, when the operations are not properly done and radically enough done, late operations come too late to be of benefit. An injured brain, lying in a closed cavity, is very susceptible to infection.

The primary factors threatening life are (1) pressure, (2) infection. The pressure may be general or local and may result from hemorrhage or from foreign body or from depressed bone fragments. The infection results from bacteria carried into the brain by foreign bodies, which may be bullet, clothing, hair, etc. Therefore, the thorough removal of all foreign bodies and provision for drainage of the wound secretion are imperative.

When, however, we undertake early operation, there arises always the difficult question: Where shall we operate? For this there is no accurate answer. It depends upon how one can arrange the field facilities. In any case, these operations can only be undertaken (1) where there is possibility for complete asepsis, (2) where assistance of the X ray can be had, and (3) where the operated patients can lie quietly for a long time after operation. Naturally, it is not always pos-

sible in the very severe cases, when life is threatened by hemorrhage, to wait for ideal surroundings, but in these cases the operation should not be more than a revision of the wound and provision for drainage and patient should be immediately sent to a location where circumstances for operation and a long post-operative treatment are to be had.

The following points are to be observed in operation: Hair must be removed, the scalp well cleansed with benzine, and tincture of iodine applied to the wound and the surrounding territory. It is probably better practice to excise the edges of the wound, particularly if ragged or dirty. Before the operation, give morphine and, if possible, carry out the operation under local anesthesia of 1 per cent novocaine. General anesthesia in severe brain injuries is contraindicated. Careful hemostasis must be secured, either by ligature or by tamponnade. Sufficient bone should be removed to allow of thorough exploration of the torn dura. Removal or elevation of all bone splinters or depressed fragments, blood clots, should be carried out. Fragments of brain should also be carefully removed, as they decompose and predispose to infection. With the gloved finger, the injured brain should be very carefully explored and very **often foreign bodies can be felt and removed.** The wounds should be left wide open or at least only partially sutured, with adequate provision for drainage.

The postoperative treatment is very important; and transportation should not be undertaken for a long time, because prolapse and infection are much more apt to follow. Primary plastic operations must not be done on account of the necessity for drainage and the danger of infection. Three or four grams of urotropin should be given daily as a prophylactic against encephalitis and meningitis.

One must be very careful in the prognosis. Gunshots of the skull must be given a much more serious prognosis than in the skull fractures seen in peace time. The reason for this lies in the much greater comminution of the bone and in the much greater injury of the brain, due to the high velocity of projectiles. The prognosis depends upon the following factors:

- (1) Upon the distance and the force of the projectile. At near distances the explosive force of the projectile, especially in through-and-through wounds, is very great and leads usually to rapid death.
- (2) Upon the amount of brain tissue injured.
- (3) Upon the location of brain tissue injured.
- (4) Upon the size of the wound in the dura, upon which in large measure the likelihood of infection depends.
- (5) Upon the character of the gunshot injury. Through-and-through gunshots and gunshots in which the projectile remains, are naturally much more dangerous than a tangential injury.
- (6) Upon the character of the projectile. Bullets offer the best prognosis; shell fragments, the worst prognosis.
- (7) Upon the time of operation. The earlier the operation, the better the prognosis.

It should be remembered, however, that even after complete healing of the wound the prognosis may still be disturbed on account of later complications, such as brain abscess, which may develop after many months, or epilepsy, and the paralyses. Nervous and psychic disturbances also develop in all kinds and degrees. Brain abscesses may develop weeks or months after the primary injury on account of infection in injured and bruised brain substance and around foreign bodies, bone fragments, etc. The differentiation from encephalitis is not always possible during the first 8 or 10 days, but encephalitis seldom develops after the first three weeks.

The prognosis in brain abscesses is not generally good, but the abscesses are usually superficial and can be easily drained, and on this account offer better prognosis than brain abscesses secondary to ear diseases as seen in peace time. It should not be forgotten that the patient, long after apparent healing, may still develop a brain abscess around a foreign body, and great care must be exercised in the discharge of these patients from observation and in their reassignment to active duty at the front.

Encephalitis is one of the most usual complications leading to death. Usually it occurs early and is difficult to diagnose from meningitis, in the majority of cases progressive, and ends fatally. Meningitis may be diffuse, extending directly from the injury, or may develop at the base. As soon as there is suspicion of meningitis, spinal puncture should be done, and if organisms are cultivated the wound should be widely opened and drained. Laminectomy may be of value. Urotropin should be given in larger doses than the usual prophylactic doses.

Brain prolapse is the expression of an inflammatory process in the brain—meningitis, encephalitis, brain abscess, or their combination. It has been observed that a prolapse may appear because of the too early transportation of patient after operation. The treatment of prolapse of the brain should be expectant. Aseptic dressings, without pressure, should be applied. In every case, as soon as the cause disappears, the prolapse will regress. If the prolapse does not disappear under expectant treatment, it means that there is "trouble" in the depths.

Axhausen, G.: Technique of Cranioplasty (Zur Technik der Schädelplastik). *Arch. f. Klin. Chir.* Bd. 107. Hft. IV, April, 1916.

The vast majority of surgeons agree that the gunshots involving skull and brain are better handled by primary operation. It is also recognized that after complete wound healing the condition of the patient, on account of nervous disturbances, does not return to an ideal state of health. Very often, at the site of injury, there is to be found a large, tender scar, through the thin, middle portion of which pulsation of the brain is to be seen, a condition known as the "pulsating skull defect." We know, further, that on

account of the very poor osteogenetic capacity of the bone of the skull, the spontaneous closure even of a small defect does not follow. In all the cases operated by Axhausen, even in the oldest, there was no attempt at formation of new bone and the margins of the defect were almost as sharp as when primary operation was completed. In order to secure complete restoration to a normal condition, a secondary filling of the defect is necessary. It is obvious in those cases in which the symptoms suggest the possibility of an infectious process within the skull, no plastic operation should be undertaken.

Axhausen considers two possibilities, the one of the flap-plastic, after Müller-König, with variations, and the free transplantation. In free transplantation, various transplantation materials are at our disposal, but there are really very few suitable materials. Certainly the most suitable of all is new bone—and bone from the patient himself. Von Eiselberg declares that the Müller-König plastic, or a variation, stands in the first line. Axhausen differs from this and bases his opinion upon many cases which he has observed in Kiel and in Berlin. During the last year, he has operated 28 cases, and he is convinced that the best method of closing defects of the skull is by the use of the free autoplasic operation. The Müller-König plastic is technically very difficult. Anyone who has repeatedly attempted the operation must admit that to secure a flap (in which the plate of bone with neighboring scalp is attached) is not at all simple. When it is successful, one may secure a thin plate made up of many broken pieces, and with this one must be satisfied. The connection between the bone plates and the covering skin is very loose, and it is impossible without loosening the scalp from the plate of bone to secure an accurate coaptation of the transplant into the bony defect. On this account, it is not possible to secure an immediate union between the implanted bone plate and the surrounding skull. On account of the poor osteogenetic capacity of the skull, it is a long time before these flaps are healed tightly. Then there is an objection for cosmetic reasons. Not only is the scar not diminished, but the throwing over of the flap makes a larger and more ugly scar, which often calls for a new operation to secure healing. Either this must be closed by second flap or by skin graft, and such scars upon the hairy scalp are very conspicuous.

Removal of a plate of bone from the anterior surface of the tibia is extremely simple. It is possible to make the plate of bone correspond accurately to the defect to be bridged and it can be made of the desired thickness. One needs no special instruments. Axhausen has used the ordinary, broad, sharp carpenter's chisel. In case the defect should be so large as not to be covered by the width of plate secured from the tibia, two pieces can be implanted alongside each other. The plate of bone from the tibia should be so implanted that it is held firmly in the defect, and it is of much assistance in freshening the defect mar-

gins to make a right-angled or a trapezoid form. Free union usually occurs in 14 days. When the transplanted portion of bone is properly inserted, no periosteal sutures for fixation are necessary.

In these 27 cases it was possible to excise the old scar and by dissecting the margins free to carry out direct suture of the skin margins above the transplanted flap. On account of the very rich circulation of the skin of the scalp it is possible to do what should not be done in other locations, and that is to bring wound edges together with some tension. There was never any difficulty caused by taking bone from the tibia: patients did not complain, and particularly it should be noticed that there were never any fractures.

Axhausen always took particular care that the periosteal side of the transplant lay outward, so that the suture line did not lie upon the bare bone but upon the overlying periosteum. The chiseled side of the bone graft lay toward the brain. This surface was fairly smooth, and Axhausen did not observe any excessive scar or callus formation. He recommends excision of as much of scar upon the brain as possible, so that there is distinct pulsation before applying the graft.

Epileptiform seizures at the close of the operation were not observed. It was twice possible, through operation, to secure a cessation of epileptic attacks. In two cases, immediately after the operation, there were symptoms of paralysis—in one of the instances combined with aphasia—but this gradually disappeared.

The question as to the proper time to undertake operation is fairly settled. In general, one must not undertake plastic operations on the skull too early. One should distinguish the cases in which there is simply injury of bone without cranial symptoms from those cases in which there are cranial symptoms, such as paralysis, aphasia, etc. The cases without cerebral symptoms may be operated slightly earlier than the others. In those without cranial symptoms operation should be done not before several weeks after the wound is perfectly healed. Those in which there is disturbance because of the brain pulsations may require operation earlier. Those patients with cerebral complications should not be operated until the neurologist has examined repeatedly and offered some opinion as to the character and the duration of the symptoms, and then operation should be postponed a few months longer. With increasing paralytic symptoms, also where there is the slightest suspicion of brain abscess, it is obvious that no plastic operation should be undertaken. Axhausen regards epilepsy as a particular indication for early operation.

Axhausen operates under general anesthesia. He objects to local anesthesia in all plastic operations, because anemia is induced, and after this secondary hemorrhage may occur which will separate the transplant from its surroundings. In young, healthy soldiers he does not see that general

anesthesia has any danger. The typical operation he describes as follows:

Excision of the existing scar in healthy skin. Deepening of the incision to the bone, which should lie outside the palpable defect. Pushing back of the surrounding skin with the pericranium sufficiently to give a good exposure of the defect. The defect margins should then be freed with the periosteal elevator, first from the overlying tissues and then from the tissues lying along the inner surface. There should be exposure of the whole extent of the defect; also in the angles. The defect should then be smoothed and whatever scar is present upon the brain should be excised. The scar should not be excised en masse, but one layer after another until pulsation of the brain is seen. Axhausen prefers leaving a very thin layer of scar to act as membrane between the brain and the transplanted bone. The margins should then be freshened with the rongeur and, preferably, the defect should be made into a right-angled or a trapezoid form. Then the skin flaps should be prepared for resuturing. In case there is too much tension, a parallel incision can be made down to the skull an inch or two away from the defect.

In taking the bone from the tibia, one makes a convex, curved incision over the anterior surface of the tibia immediately below the tuberosity, with the base of the flap lying along the course of the tibia. The flap is then thrown back, exposing the periosteum. The periosteum is then incised to the bone, somewhat more periosteum being taken than bone. Chisel is then used and driven forward with the hammer so as to remove a thin shell of bone. The chisel should be sufficiently wide, if possible, to permit the entire bone transplant being taken in one piece. A sponge should be held lightly against the piece of bone being removed, because sometimes it jumps out and is lost. While the assistant closes the wound in the tibia, the bone transplant should be trimmed to the proper shape and size and fitted accurately into the defect; should be pressed firmly into place, and the scalp closed with interrupted sutures.

Perls, J.: Symptomatology and Treatment of Gunshot Injuries of the Skull (Beitrag zur Symptomatologie und Therapie der Schädelgeschüsse). *Beiträge zur Klinischen Chirurgie. (Kriegschirurgisches Heft XXXIII.)* March, 1917, p. 435.

Perls's article is based upon his experience in the treatment of 42 cases of skull injuries in Munich, most of which have been first treated in field hospitals and sent to him several weeks after their injury with wounds clean and healing, or completely healed. From these 42 cases, 36 skull plastics were made.

The local findings in these healed cases of skull injuries were typical. He found a thin scar in the region of which a bone defect was present and the scar distinctly pulsated

with bulging on coughing. The size of the defects in the bone was up to as large as the palm of the hand. He quotes Reich, who describes the scar as follows: Adhesions bind the superficial surface of the brain with the margins of the bone defect and thus close off the subdural space; on this account the free suspension of the brain in the arachnoidal fluid is interfered with, so that by every pulsation and bulging there is irritation of brain, and this he believes leads to the development of traumatic epilepsy. Beneath the superficial scar lies a characteristic layer of edematous tissue, not a typical cyst, but resulting, evidently, from interference with the lymph drainage and disturbance of circulation. This cystic layer is not permanent and becomes further organized as a scar, which becomes a typical scar cyst.

Perls's remarks that although several histories showed that there had been an unusually large loss of brain substance, he never found beneath the scar a large defect resulting from the loss of brain substance. He explains this by "an inner brain prolapse." By this he means that the loss is compensated for by a displacement of brain substance into the defect, possibly on account of increase of ventricular fluids.

He saw at operation very often an increase of fluid and occasionally a communication with the ventricle. In one case there was pronounced discharge of fluid for six days after operation, and at autopsy a wide communication with the lateral ventricle was found.

The scar in the brain itself does not lend itself to operative attack, because naturally a fresh scar would form.

The question as to whether ganglion cells really heal he does not attempt to decide, but is of the opinion that function is taken over by other cells. The resorption of diseased brain tissue takes place slowly and extends possibly over a period of years.

Epilepsy developed in 6 of his 53 cases, and he believes that as time goes by a greater number will develop. Perls does not believe that increased pressure is responsible for epilepsy, but rather that epilepsy results in increased pressure, and he differs from Wilms, who believes that epilepsy may result directly from the increase of pressure due to a plastic operation for covering a defect. He believes that the epilepsy is due to the adhesions at the site of the scar and that the disturbance of the brain by each pulsation, by straining, by coughing, bending, makes continuous irritation on the brain, and that epilepsy results from this. Localized spasms or convulsions he saw only when the injuries were in the motor cortex. In the vast majority of cases convulsions were general.

In setting indications for operation in skull injuries with resulting defects he advises plastic operations in all large defects, and in those cases where there is distinct pulsation and bulging of the scar by coughing, etc., and in every case where the location of the defect exposes it to danger. As

to the question of whether skull plastics are indicated in epilepsy, he believes that operation should be done.

Perls repairs the defect in the bone by the method of Garre; U-shaped incision, throwing back the scalp, when the margins of the defect are freshened; and then periosteal flaps marked out, with the base near the defect, the periosteum reflected slightly; and then, with a flat chisel, a bone plate is loosened, and this bone-periosteum flap is simply turned over, with the periosteum toward the brain, in order to form an artificial dura and prevent adhesions between the bone and the brain scar. He recommends local anesthesia and emphasizes the necessity for careful hemostasis. The bone flap should consist only of the outer table, and operation should not be undertaken in any case earlier than six weeks after complete healing of the wound.

In most cases the headache, the dizziness, etc., were relieved after the defects had been repaired, and the epilepsy was relieved in half the cases, although Perls admits that epilepsy may possibly recur.

Perls also emphasizes the fact that the treatment of skull injuries does not end when the defects have been repaired. Through systematic exercises and education the patient must be brought again into the very best mental and physical condition, so that the injured portion of brain may have its functions taken over by the uninjured portion. This can best be done, not in a surgical hospital, but in a specially organized institution with special teachers and doctors. He remarks further that the later educational measures are to be undertaken with the idea that the brains to be dealt with are not diseased brains, such as fill institutions in peace times, but rather "crippled brains."

Guleke, Prof.: Skull Injuries (Schädelerschüsse). *Beiträge zur Klinischen Chirurgie. (Kriegschirurgisches Heft XIV.)* March, 1916.

Guleke takes up the question, in which cases of gunshots of the skull operative treatment should be instituted. He believes that since severe injuries may appear as simple injuries of the soft parts and a severe injury may not be recognized, it is a principle not to be departed from to explore every injury to determine the degree of bone injury. One is very often surprised at the unexpected degree of comminution of the skull and at the extent of laceration of the brain. All depressed and loose pieces of bone must be removed, the opening in the bone must be sufficiently large to disclose the extent of the injury to the dura, and the dura must be opened sufficiently wide to expose the injured portion of the brain.

Guleke is also an enthusiastic supporter of the principle of primary operation. By primary operation, he means operation at the earliest possible time where localization of foreign bodies can be done with X-ray, where operation can be performed under aseptic conditions. He lays down

the rule that when possible every foreign body should be removed at this primary operation on account of the grave danger of later abscess formation, even though the patient should recover from the primary injury and operation.

The complications most to be feared are progressive encephalitis, purulent meningitis, later abscess, and epilepsy. Late development of epilepsy he has not been able to predict, because it has developed after apparently slight and superficial injuries, while in many seemingly very severe injuries it did not follow. He emphasizes very strongly the danger of permitting patients with severe injuries of the skull, particularly with extensive brain injury, to return again to the front. He favors, on the other hand, their being kept at home, and only after a long period should they be permitted again to engage in active duty.

His conclusions are as follows: (1) Every wound of the skull should be primarily operated and at least the wound edges should be excised and the bone splinters removed. (2) In through and through gunshots and in certain cases where the projectile remains in the brain, one may postpone operation, but in the vast majority of cases primary operation should be undertaken. (3) Operation should be made early, as soon as asepsis can be secured, because delayed operation increases both the immediate operative mortality and the late complications. (4) After the wound is enlarged, sufficient drainage is the most important part of the after-treatment. An early closure of the wound flaps is to be guarded against on account of the danger of retention, abscess, cyst formation, etc. (5) Primary suture and, above all, primary plastic operations are to be avoided. (6) Brain prolapse requires only expectant treatment, unless it increases, or is indicative of deep abscess. In these cases the skull openings should be enlarged and the abscesses in the depths should be drained. (7) The number of late complications is very great and the prognosis in gunshots of the skull can, therefore, not be too cautious.

Joseph, E.: *Operative Treatment of Fresh Gunshot Injuries of the Skull* (Die Operative Behandlung Frischer Schädelschüsse). *Beit. z. Klin. Chir. Band 7. (Kriegs chirurgisches Heft XXXIII.)* March, 1917, p. 452.

Joseph says that in making diagnosis and in deciding upon operative indications for fresh skull injuries one must pay particular attention to the character of the wound and to the clinical symptoms. Especially is this true since toward the front lines X-ray apparatus is often not at one's disposal. After gunshots of head, patients are brought to the surgeon in varying conditions. Joseph warns particularly against carelessness in the first examinations, because a small wound may be overlooked, or a wound which appears to be very simple may be very serious. He has even seen cases in which the skull appeared to be intact, or with

nothing more than a fissure, when in reality the inner table was much comminuted and the brain severely injured. So, also, the absence of cerebral symptoms does not warrant, in all cases, considering the wound as harmless.

Joseph advises when possible that patients be transported away from the front for operation, and that operations should not be undertaken in the dressing stations or front hospitals unless there is X-ray apparatus and unless they are in a position to keep patients lying quietly for a long time after operation. He has seen much harm done in transporting patients immediately after operation, and he believes operation for gunshot of the head should be done in front hospitals only when the life is directly threatened by hemorrhage or pressure. In all other cases patient should be transported well to the rear, where X-ray plates can be made and where there are assurances of a long rest in bed after operation.

When operation must be done he recommends that the bone edges be smoothed and that such fragments of foreign body or bone fragments as can be easily located be removed and that the wound be left wide open. In Joseph's experience an incomplete first operation is worse than no operation, and in all the cases in which incomplete first operation was followed by a second operation patients died from infection. Insertion of drains into the brain itself is advised against, because infection may be led in. All drains should be removed within two or three days.

In discussing prolapse of the brain Joseph says that in his experience the skin edges gradually covered over the prolapsed brain, and that through contraction of scar tissue was gradually pushed back within the skull.

Joseph operated 49 gunshots of the skull. Of these, 21 died. As causes of death he ascribes infectious encephalitis and meningitis, also tetanus in two instances.

Exigencies of time have prevented including Italian literature. The following abstract, however, is included because it represents opinion expressed after the late Italian drive (August, 1917) and furnishes highly interesting mortality statistics:

Maccabruni, F.: *Treatment of Gunshot Injuries of the Head* (Trattamento delle ferite al cranio.) *Pensiero medico*, Milano, 1917, VII, pp. 286, 313.

Maccabruni's hospital usually received the wounded men a few hours after the wound was inflicted, as they were directly transported from the front by automobile. Many of these men arrived still under shock produced either by psychical trauma of the battle or by that of the wound.

Among these he had occasion to perform 44 operations on the head from August, 1916, to March, 1917. Most often

it was an "atypical craniectomy," but sometimes he had to perform a true craniectomy.

Of 44 cases operated upon 12 showed no lesion of the dura mater, in 32 cases there was a lesion of the meninges and of the brain; in 12 cases there was retention of the projectile, in the other 32 this was not the case. Almost all wounds ended blind; some were tangential; only 3 had a complete canal, and of these 1 was caused by gunshot, the other 2 by shrapnel bullets. With regard to the region injured, the wound of entrance was 10 times in the frontal region, 24 in the parietal region, 3 in the temporal region, and 7 in the occipital region.

As to the injuring agent, the wounds were divided as follows: Twenty-five from fragments of shells, 1 from a stone projected by a shell, 8 from shrapnel bullets, 6 from gunshot projectiles, 2 from bombs, 2 from the explosion of a mine. Maccabruni draws the following *conclusions*:

1. Systematic intervention in all penetrating wounds is necessary; only a very few cases make an exception to this rule.

2. The operations should be performed immediately. The sooner the intervention the better the probability of good results.

3. It is advisable to examine the nervous system in every case, avoiding tiring the patient.

4. The radiological examination should in almost all cases precede the operation to obtain as exact a location as possible of the intracerebral projectile.

5. The osseous defect should be reduced to a minimum; it should be a few millimeters larger than the meningeal lesion.

6. All bony fragments projected into the brain should be extracted; the epidural space should also be carefully explored.

7. Deep-lying projectiles should be early extracted whenever their removal is possible without producing greater damage.

8. In cases of vast wounds with tearing of brain substance drainage with gauze is extremely useful.

9. In all cases where the wound is probably infected, i. e., in the great majority of cases, the suture of the skin of the scalp should not be completely carried out.

10. The suture of the dura mater should be reduced to a minimum.

11. In lesions of the large sinuses, when hemorrhage can not be stopped with simply tamponing, it is best to use a suture, limiting the use of forcipressure to cases of extreme gravity and urgency.

12. Bandaging should be renewed as rarely as possible.

13. The rules of the most scrupulous asepsis should be followed in bandaging as well as in the operation.

14. The small cerebral hernias are reduced with compressive treatment; in large hernias all attempts at reduction should be omitted.

15. If the existence of a cerebral abscess has been diagnosed it should at once be opened and drained.

16. The nursing of the patients should be most careful.

17. The patients should not be removed until a complete surgical cure.

18. The greater part of the deaths was due to the lesion itself; less frequently to meningoencephalitis, suppurating cerebral hernia, and cerebral abscess.

Cases of opening of a ventricle, of hydrorrhea, ependymitis, and consecutive encephalitis were almost always fatal.

Of 44 cases operated upon 16 died. All cases of death belong to penetrating wounds with lesion of the dura mater and protrusion of cerebral substance. No case of penetrating wound of the head without infection of the meninges died.

CHAPTER II.

SURGERY OF THE SPINE, SPINAL CORD, AND ITS MEMBRANES.

(Parts 1-5 from Diseases of the Spinal Cord and Its Meninges, by CHAS. A. ELSBERG, M. D. Published by W. B. Saunders Company.)

PART 1.

THE SURGICAL ANATOMY OF THE VERTEBRAL COLUMN AND SPINAL CORD.

THE VERTEBRAL COLUMN.

The treatment of diseases of the spinal cord will often require the exposure of the cord by removal of the spinous processes and laminae of one or of a number of vertebrae. A thorough knowledge of the structure of the bones which make up the vertebral column and of the ligaments and muscles which bind them together is therefore necessary. For a detailed account of these structures the reader is referred to text-books of anatomy. In the following, reference will be made to a few facts of practical importance.

In the cervical and upper dorsal regions the vertebrae are smaller than in the other parts of the spinal column. The spinous processes of the cervical vertebrae are bifid at their tips, but the vertebrae do not fit closely upon one another and are rather freely movable the one upon the other. This makes the removal of spinous processes and laminae the most easy in the cervical region. In the dorsal vertebrae the laminae and spines overlap and the vertebrae are more fixed upon each other. The spinous processes point markedly downward, so that the distance between the tips of the spinous processes and corresponding segments of the cord is greater than in the cervical region. In the lower dorsal and lumbar vertebrae the spinous processes and laminae are thick and short; the spines point directly backward and are deeply placed between thick muscles. In this region the exposure of the dura in the operation of laminectomy is a more tedious

procedure; the thick, short laminae have to be removed to the transverse and sometimes into the articular processes before a wide exposure of the field of operation is obtained.

The dorsal and lumbar vertebrae are only slightly movable upon each other. When a fracture of the vertebrae occurs in this region, the injury to the spinal cord is not of necessity a great one. In the cervical region, however, the great mobility of the vertebrae upon each other will allow of much dislocation, so that severe crushing injuries with or without fracture are of more frequent occurrence.

THE SPINAL CORD AND NERVE ROOTS.

The spinal cord is about 45 cm. long: it extends from the margin of the foramen magnum to the lower part of the body of the first lumbar vertebra. At its lower end it tapers conically (the *conus medullaris*) to end in a slender filament (the *filum terminale*). The lower end of the *conus* may extend only to the twelfth dorsal or as low as the middle of the body of the second lumbar vertebra.

In the fetus the cord extends to the lower end of the spinal canal. After the third month the canal grows in length more rapidly than the spinal cord, so that at birth the tip of the *conus* lies at the level of the third lumbar vertebra. The changes in the relationship between the spinal canal and the lower end of the cord and the nerves of the *cauda equina* in the fetus and in post-natal life have an important bearing upon the symptoms of some pathological conditions.

On section the cord is almost circular, being, however, slightly flattened from before backward. The cervical and lumbar enlargements are almost entirely due to an expansion in a transverse direction. The cervical enlargement extends from the upper part of the cord to the level of the body of the second thoracic vertebra, while the lumbar enlargement begins at the tenth and is largest opposite the twelfth thoracic vertebra. The enlargements of the spinal cord are related to the large nerves which supply the upper and lower limbs. The relative size of the different parts of the spinal cord must be well understood for the proper recognition of increase in size due to intramedullary fluid or tumors, or decrease in size due to sclerotic changes.

Somewhat arbitrarily, the cord is divided into segments, each portion which corresponds to the attachments of a pair of spinal nerve roots being termed a segment. The nerve roots are, therefore, the guides to the segments of the cord, the boundaries of each segment corresponding to a horizontal plane through the cord midway between two adjacent nerve roots. There are thirty-one pairs of spinal nerves—eight cervical, twelve dorsal or thoracic, five lumbar, five sacral, one coccygeal. The first pair of cervical nerves emerge from

the vertebral canal between the occipital bone and the atlas; the first to eighth cervical roots are named after the lower of the two vertebræ which form the intervertebral foramen of exit of the nerve. The eighth cervical nerve roots emerge from the foramen between the seventh cervical and first thoracic vertebræ. In the thoracic, lumbar, and sacral areas the spinal nerves are named after the upper of the two vertebræ which form the corresponding intervertebral foramen.

Each spinal nerve is formed by the coalescence of two roots which spring from the lateral aspects of the cord, the anterior or motor root which originates from the anterolateral groove and the posterior or sensory root from the posterolateral groove. The anterior and posterior roots perforate the dural sheath separately with a thin septum of dura mater between them. In the cervical region the nerve bundles remain distinct until they have passed through the dura. The bundles are spread out like a fan, the broadest part being at the cord. At their origin the bundles are spread out so as to occupy 1 to 2 cm. of the cord; between their origin and the dura they lie closer together, forming a layer 1 to 1½ cm. in breadth; at the dural opening the nerve bundles are still distinct. In the dorsal and lumbar regions the arrangement is different from that just described; the separate bundles soon unite to form one bundle which passes outward to the dural opening.

From this arrangement it is clear that in the cervical region a tumor may, for a long time, make pressure upon only a few of the bundles which go to make up a posterior or anterior root. In the dorsal and lumbar regions the nerve bundles are united into one nerve near the cord; a tumor in these regions may press upon the whole nerve root from the very beginning of its growth. Clinical experience agrees with these anatomical facts; the earliest symptoms of pressure upon a cervical nerve root are usually confined to a small area of distribution, one or two fingers, for example, while in the dorsal or lumbar region the classical root symptoms extend over an entire root area. It is more exact, therefore, to distinguish between "root bundle" and "root" symptoms, and this distinction should be of clinical value.

There are marked differences between the course of the spinal roots at different levels. In the cervical and upper dorsal regions the nerve bundles unite to form the posterior root which passes out of the dural sac at almost a right angle to the cord. The root then perforates the dura and enters the posterior ganglion. From the ganglion each root passes outward with a slight inclination upward.

From the eighth cervical to the middorsal regions the course of the posterior roots is different. Each root has an inclination downward until it nears the dura: it bends upward at an angle just as it per-

forates the dura. In the middorsal region this angle is often very acute— 40° to 45° . Beyond the ganglion each posterior root passes markedly upward before it divides into its anterior and posterior branches.

In the lower dorsal and lumbar regions the posterior nerve roots pass downward and outward and perforate the dura; beyond the ganglia the direction remains unchanged until the nerve roots divide into their anterior and posterior branches. The course of the anterior roots corresponds to those of the posterior roots.

Taking into account the peculiar course of the nerve roots just mentioned, and the sensitive dura, it is easy to understand why a small metastatic focus of malignant disease in the posterior and lateral part of the body of a vertebra may cause those agonizing root pains from which the patients suffer. It is clear that only a slight inflammatory process near the dural opening may be responsible for the occurrence of marked root symptoms.

It is probable that the movements of the vertebral column (bending backward and forward) will increase an existing pressure upon any of the lower dorsal and lumbar posterior roots, because these can not yield as easily as the upper dorsal roots. Root symptoms in the lower dorsal and upper lumbar regions should become much intensified with forward and backward movements of the vertebral column. To a less degree this must also be the case in the cervical region, although here the bundles of the posterior roots are spread over such a large area that all are seldom pressed upon at the same time. Lateral movements of the spine are apt to increase a root pain on the opposite side and to lessen a root pain on the same side to which the spine is bent. These facts probably have an important bearing upon the occurrence of rigidity of the spine, which is found in patients who have a tumor in the lower dorsal and upper lumbar regions and also in the cervical cord.

In general, the cervical nerves pass outward through the intervertebral foramina at almost a right angle to the long axis of the cord; the lower the level, however, the more is the downward slope, so that the fifth lumbar pair emerge six vertebræ lower than the level of their origin. The lumbar and sacral nerves descend in almost parallel bundles to form the cauda equina, and conceal the delicate filum terminale. The arrangement of the nerves of the cauda equina is such that the outermost bundles correspond to the uppermost nerves.

The relation between the spines of the vertebræ and the sites of origin of the nerve roots from the cord is subject to considerable variation. This is especially the case in the thoracic region, where

some of the nerve roots show variations in their site of origin extending over a distance covered by as many as three spinous processes.

THE SPINAL MEMBRANES.

The spinal dura mater forms a loose sheath around the cord and the cauda equina and is loosely connected by areolar tissue to the periosteum of the vertebræ. On each side are the double openings for the roots of each spinal nerve, a tubular prolongation of the dura passing over the nerves for a short distance. In contact with the smooth inner surface of the dura, but not adherent to it, is the arachnoid. This is a delicate membrane which invests the cord, being separated from it by considerable fluid in the subarachnoid space.

In addition to other functions the fluid acts as a buffer to support the spinal cord and to protect it from injury.

The subarachnoid space is incompletely divided into anterior and posterior compartments by the dentate ligaments.

The pia mater is intimately adherent to the cord and forms its sheath or neurilemma. Pia mater, arachnoid and dura mater are continuous over the spinal roots, so as to form a sheath for them as they pass outward to the intervertebral foramina.

From each lateral surface of the cord a narrow fibrous band, the *ligamentum denticulatum* or dentate ligament, extends from the pia to the dura throughout the entire length of the spinal cord. It separates the anterior from the posterior roots and contributes to the support of the cord. On each side of the cord the ligament extends from the foramen magnum to the level of the first lumbar vertebra. From its attachment to the cord, each ligament extends outward and is attached to the inner surface of the dura by numerous dentations or slips. It is due to this ligament that a tumor which grows on the anterolateral or posterolateral aspect of the cord may press upon only anterior or posterior roots for a long time, and thus give only anterior or posterior root symptoms before the appearance of symptoms of pressure upon the cord itself.

The dentate ligament ends below, at the level of the first lumbar vertebra, in a fork-shaped extremity. The outer prong of the fork is usually about 1 cm. long, and is attached by its end to the inner surface of the dura. Sometimes this prong is 3 to 4 cm. long. The inner prong of the fork is attached to the pia on the lateral aspect of the cord and is prolonged downward along the side of the conus to its tip. The first lumbar posterior root rests upon this fork so that the "fork" may be used as an anatomical landmark for the

identification of the first lumbar root. The posterior roots of the lumbar and sacral nerves are dorsally placed with reference to the fibrous band on the side of the lumbosacral cord and conus derived from the dentate ligament, and can be raised up on a probe. At their origin from the lumbosacral cord the posterior roots lie close together, but when they are raised up with a probe the separate roots can often be recognized. If one begins to count from the posterior root which lies on the fork of the dentate ligament, which is the first lumbar, one can often identify each posterior root.

THE RELATION OF THE SEGMENTS OF THE CORD TO THE VERTEBRÆ.

The relations of the different segments of the spinal cord and of the nerve roots to the spinous processes of the vertebræ can be expressed as follows: In the uppermost cervical region, the origin of the nerve roots from the cord is on the same level as their point of exit from the spinal canal; the lower the nerve root, the greater the distance between its point of origin from the cord and its point of exit from the spinal canal.

In general, the lower boundary of the cervical cord (the level of the eighth cervical nerve) corresponds to the interspace between the fifth and sixth cervical spinous processes; the twelfth dorsal segment lies about opposite the ninth dorsal spine; the fifth lumbar segment corresponds to the twelfth dorsal spine. The segments of the spinal cord, therefore, lie on a higher level than the corresponding vertebræ. The fourth cervical segment lies about opposite the third cervical spine; the fourth dorsal opposite the second dorsal spine; the eighth dorsal opposite the fifth dorsal spine; the twelfth dorsal opposite the ninth dorsal spine; the second lumbar on the level of the tenth dorsal spine; the sacral segments opposite the twelfth dorsal and first lumbar spines. It must be well understood that the lower the level of the segment in question, the greater the distance between it and the correspondingly named spinous process.

THE BLOOD VESSELS AND THE MAIN FIBER TRACTS OF THE CORD.

A superficial account of the anatomy of the spinal cord and the course of its conducting fibers would carry me far beyond the bounds of what is necessary in this volume. Presupposing, therefore, that the reader has a good knowledge of this subject or will obtain detailed information from larger textbooks of anatomy and physiology. I shall mention only a few facts of practical importance.

The spinal cord is incompletely divided into two halves by the fissures which pass in from the anterior and posterior surfaces of the

cord. The anterior fissure is wider and shorter than the posterior one and reaches to the white commissure. It contains a fold of pia mater and many blood vessels. The posterior fissure or septum also serves to conduct blood vessels to the substance of the cord. When an incision is made in this septum, considerable oozing of blood is apt to occur, and therefore it is advisable to make the incision rather a little to one side of the septum. There are shallow furrows on each side of the cord in front and behind, corresponding to the lines of attachment of the anterior and posterior roots. Other grooves mark off the mediodorsal or Goll's column and the laterodorsal or Burdach's column. The gray matter occupies the more central parts of the cord, and appears in the form of two irregularly crescentic portions on each side united across the median line by the gray commissure.

The blood supply of the spinal cord is derived from the anterior and posterior spinal arteries—branches of the vertebral arteries. The posterior spinal vessels are of special importance because they are the ones that are most often injured in spinal fractures, and because of their position on the posterior aspect of the cord they are in the operative field during laminectomy. The posterior spinal arteries run down on each side of the posterior surface of the cord in front of the origins of the posterior nerve roots. They are more or less tortuous and give off numerous branches which perforate the cord and the posterior median septum. Branches of the posterior spinal veins form a free anastomosis around the dorsal roots of the spinal nerves and accompany each posterior root through the opening in the dura. These branches are sometimes much enlarged so that they cause undue pressure upon the nerve roots and cause root pains. Branches of the two posterior spinal arteries anastomose with each other so that the vessels may be considered as a series of communicating loops.

The spinal conducting paths are either exogenous, which originate in cells outside of the cord, or endogenous, which originate from cells within the cord substance.

The exogenous fibers are either centrifugal or centripetal. The main fiber tracts are the following:

A. I. CENTRIFUGAL OR DESCENDING (MODIFIED FROM BING).

Name.	Origin.	Course.	Ending.
1. Corticospinal or pyramidal: (a) Direct. (b) Lateral.	Motor cortex of brain..	(a) In anterior columns..... (b) In lateral columns after decussating in medulla.	In cells of anterior horns.
2. Subcortical: (a) Rubrospinal (Monakow's bundle). (b) Thalamospinal..	Red nucleus..... Optic thalamus.....	In lateral columns after crossing. In lateral columns with rubrospinal tract after crossing. do.....	Do. Do. Do.
(c) Tectospinal..... (d) Vestibulospinal.	Roof of midbrain..... Deiter's nucleus.....	In anterior columns without crossing.	Do. Do.

II. CENTRIPETAL OR ASCENDING.

1. Short fibers from posterior roots.	Spinal ganglia from periphery.	Through marginal zone.....	In cells of anterior and posterior horns.
2. Long fibers from posterior roots.	do.....	Through posterior horns without crossing.	In Clarke's columns.
3. Long fibers.....	do.....	Through posterior columns..	In nuclei of posterior columns and medulla.

B. I. ENDOGENOUS FIBERS.

1. Spinocerebellar: (a) Dorsal (Flechsig) (b) Ventral (Gower's) (c) Spinothalamic..	Cells of Clarke's column. Cells of anterior horn.. Cells of posterior horn..	Lateral column without crossing. Anterior part of lateral column partly crossed, partly uncrossed. In lateral column after crossing.	In cerebellum (vermis). Do. In optic thalamus.
2. Association tracts: (a) In anterior columns. (b) In posterior columns.			
3. Root fibers.....	Cells of anterior horn..	Peripheral nerves.	

PART 2.

THE NORMAL AND PATHOLOGICAL PHYSIOLOGY OF THE SPINAL CORD.

The spinal cord is a sensory, motor, vasomotor, and trophic organ.

1. It contains fibers for the transmission of various kinds of sensation—tactile, temperature, pain, deep muscle, and joint sense. The fibers for touch enter the cord in the posterior roots and pass upward in the posterior columns partly on the same and partly on the opposite side. The fibers from each root lie outside and somewhat superficial to those from the next lower root. They thus form a series of lamellar tracts, the fibers from each posterior root as they enter the posterior columns crowding the fibers from the next lower nerve root more toward the median line. From this it results that the fibers for tactile sensation from the lumbar and sacral nerves lie nearest the median line, the dorsal more external and posterior, and so on. When one part of the sensory pathway in the cord is interrupted other tracts may take up the transmission of sensations. Although this is probably true for all kinds of sensation, the tactile sensations especially seem to run in a bilateral path and are not so apt to be markedly affected by a purely unilateral lesion.

The fibers for temperature and pain sensation enter in the posterior roots, soon pass into the gray matter of the posterior horns, and crossing over to the other side enter the anterior part of the lateral columns and run upward to the optic thalami. The fibers for the sensations of temperature do not cross to the other side as quickly as the fibers for pain, but require from two to five segments for their complete crossing.

The fibers for deep muscle sense (postural recognition, spacial discrimination—bathysthesia of Oppenheim) in part pass up in the posterior columns; in part they enter the posterior horns, pass outward and then upward in the posterior parts of the lateral columns (lateral cerebellar tracts) to end in the cerebellum.

2. The pyramidal tracts, lying in the lateral and in the mesial parts of the anterior columns, contain the main fibers for the transmission of motor impulses to the muscles through the anterior roots. Interference with these tracts is not always followed by a complete paralysis, because there are a number of secondary tracts which contain fibers from lower centers to the motor nerve cells in the

anterior horns which can to a certain extent take up the functions of the pyramidal tract fibers. While destruction of the pyramidal tracts may not cause a complete paralysis, the destruction of the cells in the anterior horns (which are secondary centers for the muscles of the trunk and extremities) will be followed by a complete paralysis and atrophy of the muscles innervated from these cells. Muscle tone and the muscle and tendon reflexes are controlled through the pyramidal tracts and the anterior spinal roots. Muscle tone is controlled through the cells in the anterior gray horns, but these are under the influence of sensory stimuli which are continually passing to the cells from the periphery, both from the skin and from the muscles themselves. The tone of the muscles would be too great and the reflexes too active if it were not for controlling and regulating influences from higher centers which pass down to the cells in the anterior horns through the fibers in the pyramidal tracts. These considerations explain why a lesion of the pyramidal tracts is followed by hypertonus and an exaggeration of tendon reflexes, a lesion of the anterior spinal roots by a loss, and lesion of posterior spinal roots or posterior white columns by a more or less well-marked diminution in muscle tone and in the tendon reflexes.

3. The cells in the gray horns form the trophic centers for the *motor* nerve fibers which originate from them. Destruction of the cells in the anterior horns is followed by a typical Wallerian degeneration in the nerve fibers from these cells, followed by a degenerative atrophy of the muscles, so that they no longer respond in the normal manner to the electrical current. The typical "reaction of degeneration" consists of (1) a loss of contractility of the muscles when the nerves are stimulated by either the faradic or galvanic current, and (2) a loss of faradic contractility and an increased irritability to galvanism of the muscles themselves. When complete degenerative atrophy has occurred neither nerve nor muscle will respond to faradic or galvanic stimulation. The *sensory* nerves which are continued as the posterior spinal roots will similarly degenerate when they are separated from the spinal ganglia, which are the trophic centers for the peripheral sensory nerves. The central nervous system exercises a trophic influence upon all of the tissues of the body, upon the muscles and bones, as well as the skin and its adnexa. There is no satisfactory evidence that there are special trophic nerves, and most investigators of this subject have concluded that in a way not yet understood the central nervous system—brain and spinal cord—exerts this trophic influence.

4. The spinal cord also exercises a control over the vasomotor system. Lesions of the cord may result in either increased redness and heat of the skin with hyperidrosis, or in cyanosis or pallor, fall in

cutaneous temperature and anidrosis. As far as is known, the vasomotor centers lie in the anterior gray horns of the spinal cord.

From what has been stated above, we can in a general way understand the symptoms which will arise from disease or destruction of fibers or cells in the cord. We must next determine what are the functions of the different parts of the cord and of the nerve roots, and what symptoms follow the division of nerve roots or cord tracts.

The functions of the spinal roots.—Irritation of a posterior or sensory spinal root will cause pain, hyperesthesia or paresthesia over part or the entire area of distribution of the nerve root. Disease or pressure may affect only a few of the bundles that make up a posterior root; this will occur most often in the cervical region where the nerve bundles originate over a considerable length of the cord and unite only at the dural opening.

If the entire nerve root is destroyed, there may be some sensory disturbances (hypesthesia). According to the so-called "Sherrington's law," each area or zone of the skin is supplied by three spinal roots and anesthesia will occur only when three successive roots have been destroyed. In these cases all kinds of sensation—tactile, pain, thermal, deep muscle—are lost.

Division of a number of posterior roots from an extremity will cause a marked atony and ataxia of the limb. This is due both to the loss of deep muscle sense and to the destruction of the afferent part of the reflex arcs which control the tone of the muscles.

As will be shown in the chapter on spinal localization, root pains and root anesthetics are important in the localization of the level of a spinal lesion.

In the case of the *anterior* roots, the question is a more simple one. Irritation of an anterior root will be followed by muscle spasm or hypertonicity; division of one or more roots will be followed by weakness and loss of tone or paralysis of the muscle or muscles innervated by the root, followed sooner or later by atrophy of the muscles.

Results of lesions of the central gray matter.—In these cases the sensory symptoms are generally of the nature known as dissociated, i. e., there is a disturbance in the thermal and pain senses with persistence of normal or almost normal tactile sense. The disturbance in thermal sensation is usually incomplete unless parts of the lateral white columns are also affected, and warm is sometimes better felt than cold, or vice versa. At other times there may be a perversion of the temperature sense, so that cold is felt as warm, and warm as cold. The sensory disturbance may occur over large or small areas.

The motor symptoms consist of weakness or paralysis of the muscles whose centers lie within the affected area, followed by rapid degenerative atrophy.

Division of the posterior white columns.—If the posterior white column of one side alone is divided, there is no sensory disturbance at all or only some diminution of deep muscle sensibility on the same side. If both posterior columns are divided, there will be a diminution or loss of tactile and deep muscle sensation below the level of the lesion. In unilateral division of the posterior column there may also be slight disturbance in pain and thermal sensibility on the side opposite to the lesion, if the marginal root area has been injured (injury to root fibers in the marginal root zone).

Division of a lateral white column will cause a paresis or paralysis of all the muscles supplied below the level of the lesion on the same side, associated with a marked increase of tone of the muscles (spasticity) and an increase of muscle and tendon reflexes. Vasomotor disturbances are sometimes observed. The sensory changes affect the thermal and pain senses on the opposite side. Homolateral and contralateral ataxia have also been observed.

Division of one-half (hemisection) of the cord.—The symptoms which follow present a well-recognized picture known as the Brown-Séquard syndrome, from the author, who was the first to make a careful study of the symptoms. In the typical cases there are motor paralysis, superficial hyperesthesia, loss of deep muscle sense and vasomotor disturbances on the side of the lesion, and loss of tactile, pain, and temperature sense on the opposite side.

Complete transverse division of the cord is followed by a loss of all motor and sensory power below the level of the lesion, with loss of superficial and deep reflexes and paralysis of the bladder and rectum. Occasionally, slight reflexes will persist for a short time, but in the majority of instances the loss is immediate and complete. The symptoms of an incomplete transverse lesion of the cord will be considered in another chapter.

THE SENSITIVENESS OF THE CORD AND MEMBRANES.

Although our knowledge of this subject is still meager, investigations I have made, which substantiate the results of others, have led me to the following conclusions: The outer surface of the dura is insensitive, while its inner surface is very sensitive. When the inner surface of the dura is scratched or rubbed, a distinct pain referred to the back is complained of by the patient. The pia arachnoid and the dentate ligament are not sensitive. A slip of the ligament may be grasped with a forceps and divided with scissors without pain.

The cord tissue itself is not sensitive to pain, as far as I have been able to determine during my operations under local anesthesia and

in experiments on animals. When an incision is made in the posterior columns near the origin of the posterior roots, however, the patient will complain of a peculiar burning pain which may be referred to an extremity, although often the patient will declare that he feels pain but is unable to localize it.

The posterior spinal roots are very sensitive in their entire course. the painful sensations being referred to the periphery. In one or two instances I have gained the impression that the anterior nerve roots were not entirely insensitive, but I am in some doubt as to the correctness of these observations.

PART 3.

THE LOCALIZATION OF MOTOR, SENSORY, AND REFLEX FUNCTIONS IN THE DIFFERENT SEGMENTS OF THE SPINAL CORD.

The spinal centers for the muscles of the trunk and extremities lie in the cells of the gray matter of the cord. Most of the muscles have a plurisegmental innervation and there is no evidence that, as in the brain, distinct nuclei for single muscles or even for muscle groups exist.

With few exceptions the muscles are innervated from groups of cells which extend over several segments. Muscles with different functions may be innervated through the same anterior root (Forgue and Lannegrace, Martin, Sherrington). According to Herringham, of two muscles, both of which receive their nerve supply from cells in several segments, the one nearer the head, the one nearer the long axis of the body, or the one of the two that is more superficially placed will be supplied by the uppermost of the segments in question.

If the general facts just given be kept in mind, the localization of the centers for the muscles will be easily understood. The following table has been modified from that of Flatau (*Handbuch der Neurologie*, Vol. I, Part II, pp. 659 to 661), to which I have added the main function of each muscle or muscle group.

THE SEGMENTARY LOCALIZATION OF THE MUSCLES.

A. Muscles of the head and trunk.

I. Muscles of the back.

Trapezius.....	C 2-4.....	Rotation of scapula, raises shoulder, moves head to side.
Latissimus dorsi.....	C 6-8.....	Adducts and rotates arm inward, draws arm backward, raises ribs.
Rhomboid.....	C 4-5.....	Rotates and moves scapula backward.
Levator anguli scapulae.....	C 3-5.....	Raises angle of scapula.
Serratus posticus superior.....	D 1-4.....	Respiration.
Splenius capitis.....	C 2-8 (?).....	Rotates head and draws it to side.
Paravertebral muscles.....	C 1-8 3.....	Movements of vertebral column.
Rectus capitis major.....	C 1-2.....	} Rotate head and draw it back.
Rectus capitis minor.....	C 1.....	
Obliquus capitis superior.....	C 1.....	} Rotate head and draw it back.
Obliquus capitis inferior.....	C 2.....	

II. Muscles of neck, chest, and abdomen.

Muscles of the neck:

Platysma.....	C 3.....	Depresses lower lip and angle of mouth, wrinkles skin of neck.
Sterno-mastoid.....	C 2-3.....	Rotates and draws head to shoulder, muscle of inspiration.

A. Muscles of the head and trunk—Continued.*II. Muscles of neck, chest, and abdomen—Continued.***Muscles of the neck—Continued.**

Sterno-hyoid.....	C 1-3.....	Depress larynx and hyoid bone, control movements of thyroid cartilage.
Omo-hyoid.....	C 1-3.....	
Sterno-thyroid.....	C 1-4.....	
Thyro-hyoid.....	C 1-2.....	Flex and rotate cervical vertebral column.
Longus colli.....	C 2-8.....	
Longus capitis.....	C 1-4.....	Flexes and rotates head.
Rectus capitis anticus.....	C 1.....	
Scalenus anticus.....	C 4-7.....	Raise ribs for inspiration.
Scalenus medius.....	C 2-8.....	
Scalenus posticus.....	C 5-8.....	

Muscles of the chest:

Pectoralis major.....	C 5-6.....	Adduction, downward and forward movement of arm.
Pectoralis minor.....	C 7-8 (D1).....	
Subclavius.....	C 5-6.....	Depresses shoulder.
Serratus anticus.....	C 5-7.....	Fixes scapula.
Levatores costarum.....	C 8-D 11.....	Fix ribs.
Intercostals.....	C 2-11.....	Inspiration.
Triangularis sterni.....	D 3-4.....	Expiration.
Diaphragm.....	C 3-5.....	Respiration.

Muscles of abdomen:

Rectus abdominis.....	D 5-12.....	Constrict cavity of abdomen, assist expiration, rotate trunk, move pelvis.
Pyramidalis.....	D 12-L 1.....	
Obliquus externus.....	D 5-12.....	
Obliquus internus.....	D 8-L 1.....	
Transversalis.....	D 7-L 1.....	Moves pelvis and trunk, inspiration.
Quadratus lumborum.....	D 11-L or L 1-4.....	
Coccygeus.....	S 3-5, C.....	

B. Muscles of the extremities.*I. Muscles of the upper extremities.***(a) Shoulder:**

Deltoid.....	C 5-6.....	Abduct arm to horizontal, abduct and rotate arm outward.
Supraspinatus.....	C 5.....	
Infraspinatus.....	C 5-6.....	Rotates arm outward.
Teres minor.....	C 5.....	
Teres major.....	C (5), 6, (7).....	Rotates arm inward.
Subscapularis.....	C 5-6.....	Rotates arm inward.

(b) Arm:

Biceps.....	C 5-6.....	Flexes and supinates forearm.
Coraco-brachialis.....	C 6-7.....	Adducts forearm.
Brachialis anticus.....	C 5-6.....	Flexes forearm.
Triceps.....	C 6-7 (8).....	Extends forearm.
Subanconeus.....	C 7 (8).....	Fixation of synovial membrane.

(c) Forearm:

Pronator radii teres.....	C 6-7.....	Pronates forearm.
Flexor carpi radialis.....	C 6-7.....	Flexes and radially flexes hand.
Palmaris longus.....	C (7), 8, (D 1).....	Flexes hand.
Flexor carpi ulnaris.....	C (7), 8, (D 1).....	Flexes and ulnar flexes hand.
Flexor sublimis digitorum.....	C 7-8, D 1.....	Flexes middle phalanges, 2-5 fingers.
Flexor profundus digitorum.....	C 7-8, D 1.....	Flexes last phalanges, 2-5 fingers.
Flexor longus pollicis.....	C 6-7.....	Flexes last phalanx of thumb.
Pronator quadratus.....	C 6-8, D 1.....	Pronates forearm.
Supinator longus.....	C 5-6.....	Flexes forearm.
Extensor carpi radialis.....	C (5), 6-7.....	Extends radially, flexes hand.
Extensor communis digitorum.....	C 6-8.....	Extension of first phalanges, 2-5 fingers.
Extensor minimi digiti.....	C (6), 7-8.....	Extension of first phalanx of little finger.
Extensor carpi ulnaris.....	C (6), 7-8.....	Extension and ulnar flexion of hand.
Supinator brevis.....	C 5-7.....	Supinates forearm.
Abductor longus pollicis.....	C 6-7.....	Abducts first metacarpal.
Extensor brevis pollicis.....	C 6-7.....	Extension of first phalanx of thumb.
Extensor longus pollicis.....	C 6-7, (8).....	Abducts first metacarpal: extension of last phalanx of thumb.
Extensor proprius indicis.....	C 6-8.....	Extension of first phalanx of index-finger.

B. Muscles of the extremities—Continued.*I. Muscles of the upper extremities—Continued.**(d) Hand:*

Abductor brevis pollicis.....	C 6-7.....	Abducts first metacarpal.
Flexor brevis pollicis.....	C 6-7.....	Flexes first phalanx of thumb.
Opponens pollicis.....	C 6-7.....	Opposition of first metacarpal.
Adductor pollicis.....	C 6-7.....	Adducts first metacarpal.
Abductor minimi digiti.....	C 8, D 1.....	Abducts little finger.
Flexor brevis minimi digiti.....	C (7), 8, (D 1).....	Abducts and flexes little finger.
Opponens minimi digiti.....	C (7), 8, (D 1).....	Draws forward 5th metacarpal.
Lumbricales.....	C 7-8, (D 1).....	Abduct and adduct fingers.
Interossei.....	C 7-8, (D 1).....	Extension of 2d and 3d phalanges.

*II. Muscles of the lower extremities.**(a) Hip:*

Iliacus.....	L 2-4.....	Flexion at hip.
Psoas major.....	(D 12), L 1-3, (4).....	} Flexion at hip.
Psoas minor.....	(D 12), L 1-3, (4).....	
Gluteus maximus.....	(L 4), 5, S 1, (2).....	Extension of thigh.
Tensor fasciæ latae.....	L 4-5.....	Flexion of thigh.
Gluteus medius.....	L 4-5, S 1.....	} Abduction and internal rotation of thigh.
Gluteus minimus.....	L 4-5, S 1.....	
Piriformis.....	S 1-2.....	External rotation of thigh.
Obturator internus.....	L 5, S 1-2.....	} External rotation of thigh.
Quadratus femoris.....	L 4-5, S 1.....	

(b) Thigh:

Sartorius.....	L 2-3.....	Internal rotation of leg.
Rectus femoris.....	L 2-4.....	} Extension of leg.
Vastus medius.....	L 2-3.....	
Vastus internus.....	L 2-4.....	
Vastus externus.....	L 3-4.....	
Pectineus.....	L 2-3.....	} Adduct thigh.
Adductor longus.....	L 2-3.....	
Gracilis.....	L 2-4.....	
Adductor brevis.....	L 2-4.....	
Adductor magnus.....	L 3-4.....	} Adduction and external rotation of thigh.
Obturator externus.....	L 3-4.....	
Biceps.....	L (4), 5, S 1-2.....	} Flex leg.
Semitendinosus.....	L 4-5, S 1.....	
Semimembranosus.....	L 4-5, S 1.....	

(c) Leg:

Tibialis anticus.....	L 4, (5).....	Dorsal flexion and supination of foot.
Extensor longus digitorum.....	L 4-5, S 1.....	Extension of toes.
Peroneus tertius.....	L 5, (S 1).....	Dorsal flexion and pronation of foot.
Extensor longus pollicis.....	L 4-5 (S 1).....	Extension of large toe.
Peroneus longus.....	L 5, S 1.....	} Dorsal flexion and pronation of foot.
Peroneus brevis.....	L 5, S 1.....	
Gastrocnemius.....	L (4), 5, S 1-2.....	} Plantar flexion of foot.
Soleus.....	L (4), 5, S 1, (2).....	
Plantaris.....	(L 4-5, S 1).....	
Popliteus.....	L 4-5, S 1.....	Flexion of leg.
Tibialis posticus.....	L 5, S 1, (2).....	Adduction of foot.
Flexor longus digitorum.....	L 5, S 1-2.....	Flexion of last phalanges II to V.
Flexor longus pollicis.....	L 5, S 1-2.....	Flexion of last phalanx of large toe.

(d) Foot:

Extensor brevis pollicis.....	L 4-5, (S 1).....	Extension of large toe.
Extensor brevis digitorum.....	L 4-5, S 1.....	Extension of toes.
Abductor pollicis.....	L 5-S 1.....	} Movement of toes.
Flexor brevis pollicis.....	L 5-S 1.....	
Adductor pollicis.....	S 1-2.....	
Abductor minimi digiti.....	S 1-2.....	
Opponens minimi digiti.....	S 1-2.....	
Flexor brevis digitorum.....	L 5-S 2.....	
Lumbricales.....	S 1-2.....	
Interossei.....	S 1-2.....	

THE SEGMENT DISTRIBUTION OF SENSATION IN THE BODY.

Anatomical, physiological, and experimental investigations have led to the division of the skin of the body into a number of areas or zones (Head, Thorburn, Starr, Mackenzie, Petriën, Sherren, etc.) in relation to the different spinal segments. These areas are not distinct but overlap each other, so that each area is supplied from three spinal segments (Sherrington). This overlapping does not occur to the same extent in all parts of the body; on the chest and abdomen the zones overlap each other only partially while in the hand, for example, each area of skin is supplied by three spinal roots. It is of some practical importance to remember that the overlapping for the sensation of pain is always less than that for touch.

Notwithstanding the large amount of investigation that has been made, the exact size and shape of the areas supplied by the several spinal roots have not been determined with certainty. Probably considerable variations occur in different individuals.

4. In addition to these reflexes, the following centers must be mentioned:

1. The cilio spinal center..... C 8 to D 1
2. The center for the bladder..... S 3-4
3. The center for the rectum..... S 3-4
4. The center for the sexual organs..... S 2-3
5. The spinal respiratory center (phrenic nerve)..... C 3-5

THE CENTERS FOR THE SKIN AND TENDON REFLEXES IN THE SPINAL CORD
(ACCORDING TO BING).

Tendon reflex.	Skin reflex.	Elicited by—	Result.	Localiza- tion.
	Scapula reflex.....	Irritation of skin over scapula.	Contraction of muscles of shoulder blade.	C 5-D 1.
Biceps reflex.....		Blow on tendon of biceps..	Flexion of forearm.....	C 5-6.
Triceps reflex.....		Blow on tendon of triceps..	Extension of forearm.....	C 6-7.
Scapulo-humeral reflex.....		Blow on lower inner angle of scapula.	Adduction of arm.....	C 6-7.
Radius reflex.....		Blow on styloid process of radius.	Supination of forearm...	C 7-8.
	Palmar reflex.....	Irritation of palm of hand.	Flexion of fingers.....	C 8-D 1.
	Epigastric reflex.....	Irritation of lower part of chest.	Contraction in epigastric region.	D 7-9.
	Upper abdominal reflex.....	Irritation of skin over upper abdomen.	Contraction of abdominal muscles.	D 8-9.
	Lower abdominal reflex.....	Irritation of skin of lower abdomen.	do.....	D 10-12.
	Cremaster reflex.....	Irritation of region over adductor muscles.	Elevation of testis.....	L 1-2.
Patellar reflex.....		Blow on tendon of quadriceps.	Extension of leg.....	L 2-4.
	Gluteal reflex.....	Irritation of skin of gluteal region.	Contraction of gluteal muscles.	L 4-5.
Achilles reflex.....		Blow on Achilles tendon.	Extension of foot.....	S 1-2.
	Plantar reflex.....	Irritation of skin of sole.	Flexion of toes.....	S 1-2.
	Anal reflex.....	Pricking of perineum.....	Contraction of sphincter ani.	S 5.

PART 4.

THE SYMPTOMATOLOGY OF SPINAL DISEASE.

The symptoms of a spinal disease, whether or not that disease is amenable to surgical treatment, will depend upon the nature of the disease and the part of the spinal cord and nerve roots that have become affected. Some diseases attack particular tracts or groups of nerve cells; others are destructive in nature—cells, fibers, or entire tracts being destroyed; in still others the functions of cell groups and conducting fibers are inhibited by pressure, and in the early stages little or no actual injury occurs. It is characteristic of all of these varieties, however, that sooner or later secondary destruction—ascending or descending degeneration—occurs. The early symptoms will depend mainly upon the parts of the spinal cord first affected. Thus, diseases which cause a localized increase of pressure will often give early anterior or posterior root symptoms, either spasms or weakness or paralysis of muscles or pain or other sensory disturbances. Associated with these early symptoms there is usually more or less rigidity of the spinal column. The stiffness of the back is generally a defensive phenomenon, because the pain is generally made worse by movements of the back or by sudden shocks to the spine as occur in sneezing and coughing. The scoliosis which is often seen in spinal compression due to tumor is also to be explained as a defensive phenomenon.

After the root symptoms have existed for a certain time (and the period may extend over months or many years), symptoms referable to the cord appear. These are either weakness or paralysis of groups of muscles or of entire extremities, and sensory disturbances due to interruption of centripetal fibers in the white columns or gray matter of the cord. The progression of symptoms is sometimes very slow, extending over months or years, with frequent remissions, or it is more rapid, sensory and motor loss becoming marked and extensive, within a short time. These are soon followed by disturbances in the functions of the bladder and rectum, and, finally, with marked trophic disturbances, decubitus and edema of the extremities.

The above is the general course of symptoms when the spinal cord is subjected to a gradually increasing pressure from without. When, on the other hand, the disease originates within the cord substance itself, the progression of symptoms is usually a somewhat different one. While pain may be an early symptom, it is generally absent in

the early stages of intramedullary disease. The first symptoms are generally motor—weakness and atrophy of muscles or muscle groups, with gradual extension until more or less of one or of several extremities is affected. With these early motor symptoms there are sensory disturbances of the dissociated type, or sensation remains normal. The patients often complain of a feeling of numbness or cold in the limbs.

In the first variety of disease, i. e., in which pressure upon the cord from without occurs, the sensory and motor symptoms sooner or later have a distinct level character; in the primary intramedullary disease there may be the same evidence of a level lesion. In many cases of intramedullary disease, however, the affection spreads upward (and downward) in an irregular way, affecting parts of the various fiber tracts so that no distinct level can be recognized. If a level of the disease is observed, it may be shifted upward by the gradual advance of the disease. Similarly, in some extramedullary diseases (leptomeningitis, pachymeningitis) the level of the disease may be slowly shifted upward with the advance of the inflammatory process.

From what has been said, it is clear that the recognition of the *sequence* of the symptoms is very important for the diagnosis and correct valuation of the symptoms of spinal diseases.

1. **The sensory disturbances due to spinal disease** affect the three elementary sensations—touch, pain, and temperature. In lesions within the substance of the spinal cord, pain or temperature sense (if affected at all) is diminished or lost, but it is the entire pain or the entire temperature sense. In root lesions, on the other hand, we regularly observe the peripheral type of sensory disturbance described by Henry Head. When a posterior root lesion is suspected we must examine separately for superficial and deep pain sense disturbance, and for disturbances in the protopathic (above 45° and below 20° C.) and the epicritic (between 25° and 40° C.) temperature sense. In the peripheral nerves there is a close connection between the sensation of pressure, of painful pressure, and the power of recognition of the position of the limbs, or of passive movements, while (according to Head) in spinal disease the power of recognizing the direction and nature of passive movements and of the sense of pressure may be distinct from deep muscle sense.

The sensory symptoms are either subjective or objective. Under the first heading are included pain and paresthesia; under the second, a great variety of disturbances of sensation discovered by physical examination.

The pain of spinal disease may vary much in its intensity. Disease of or pressure on posterior spinal roots will cause typical root pains

usually referred to the periphery—down one or the other limb, to part of the chest or abdomen. The so-called intercostal neuralgia is most often a real root pain, and recent investigations have demonstrated that some, at least, of the indefinite abdominal disturbances complained of by patients are due to sensitive cutaneous areas on the abdominal wall. In the so-called “neuritis” of one of the extremities, unless its cause has been determined, we must always be on the lookout for a possible spinal root lesion. Many patients with spinal lesions, especially with intradural new growths, complain of pain in the back which is different from root pain and which is, I believe, due to irritation of the sensitive inner surface of the *dura mater*.

When paresthesiæ are complained of there is usually a “pin-and-needle sensation,” a feeling of numbness or coldness, or burning sensations. These paresthesiæ are sometimes referred to particular nerve areas, but more often are referred to the peripheral parts of extremities. In the former case their localization has considerable diagnostic value, especially when an objective sensory disturbance is found over the same area.

Objective sensory disturbances mainly consist of increase, diminution, or disappearance over definite areas of one or more of the three main forms of sensation—touch, pain, or temperature. In complete destructions of the cord at any level, there is a total loss of all sensation below the affected level. In partial lesions of the cord the sensory disturbance will depend upon the level of the lesion and the tracts affected.

A hypersensitiveness to touch, pain, or heat and cold often occurs at the level of a spinal lesion and is probably due to irritation of posterior spinal nerve roots at that level. If the function of a posterior nerve root at the level of the disease is totally interfered with, there may be an anesthesia over the area of distribution of the root, but this rarely occurs unless three roots at least are affected. Hence we may find at or above the general level of the sensory disturbances a hyperesthesia or an anesthesia which is radicular in character, and when this occurs on one side of the body only, we may be certain that the lesion—if a localized one—is on that side of the spinal cord.

The amount of disturbance of sensation below the level of the lesion will depend upon the degree of interference with fiber tracts. The amount of tactile disturbance is not apt to be as marked as that of the other sensations for reasons that have been explained elsewhere, for tactile sensation is lost only when there is extensive disease of the cord. Loss of tactile sense does, however, occur alone, although in most cases it is associated with changes in the pain and temperature senses.

Disturbances in tactile and deep muscle sensation will regularly follow a disease of both posterior white columns of the cord, but numerous cases are on record where disease of one posterior column did not cause any tactile disturbance. This can only be understood on the basis that the fibers for tactile sensation run in a bilateral path and that one tract can take up the functions of the other, if necessary. Recently, Head and Thompson have claimed that the power of discrimination between two points on the skin is diminished or lost in posterior column disease, and that if only one posterior column is affected, this diminution or loss will be found only on the affected limbs of that side. If the statements of Head and Thompson are correct (as they seem to be), the diminution or loss of the power of discrimination is a valuable diagnostic symptom.

Loss of the sense of vibration (pallanesthesia of Oppenheim) may be an early symptom of sensory disturbance in tumors, compression paraplegia, multiple sclerosis and syphilis of the spinal cord.

In the beginning of pressure upon fiber tracts of the cord there may be very slight sensory disturbance, which can be discovered only by the most careful examination. If the posterior columns are most affected, the sensory loss will involve mainly tactile (hypesthesia or anesthesia) and deep muscle sense. If the lateral columns are also affected, pain (hypalgesia or analgesia) and temperature sensation are also involved.

Usually the temperature and pain senses are equally affected; occasionally the one or the other sensation is preserved. Sometimes the recognition of cold is preserved while that of warm is lost, or vice versa; at other times there is a perversion of sensation, heat being felt as cold and cold as heat. Instead of pain, there may be a burning or itching sensation. This dysesthesia was first described by Charcot; it has often been observed in stab wounds of the cord.

When the disturbances of the pain and temperature senses are due to a lesion of the lateral white columns, an ataxia of the affected limbs is often observed (spinocerebellar tracts in lateral columns).

Marked disturbance of the pain and temperature senses with little or no affection of the tactile sense is known as "dissociation of sensations," and is especially frequent in disease of the gray matter of the cord (syringomyelia, hematomyelia, central tumors of the cord). Until recently it was believed that this dissociation of sensibility always signified central disease of the cord, but we now know that it may occur in disease of a lateral column and is not so very rare in extramedullary tumors of the cord which cause the Brown-Séquard symptom complex. When these dissociated sensibility disturbances are associated with early muscular paralyses and early wasting of special muscles or groups of muscles, the diagnosis of central disease of the cord is more probable.

Finally, the physician must never forget that severe disease of the cord may exist without any sensory disturbance; that, for example, a tumor on the anterior surface of the spinal cord may not cause any objective sensory disturbances for a long time.

2. **Interference with the pyramidal tracts** in any part of their spinal course will cause motor symptoms whose character will depend upon the amount of the interference. Thus there may ensue paresis or paralysis with or without spasticity, changes in the cutaneous muscle and tendon reflexes or disappearance of reflexes. The amount of paralysis will depend upon the degree of interference with the conducting fibers in the pyramidal tracts. Although many writers have attempted to formulate a general plan of arrangement of the fibers for the different parts of the limbs in the columns of the cord, there is, as yet, little unanimity on this subject.

Lesions of the pyramidal fibers will cause an increase in the reflexes below the level of the lesion in most (but not all) instances. The reflexes are either cutaneous, tendinous, or periosteal.

In the upper extremities the important reflexes which become exaggerated when there is a lesion in the pyramidal tracts above the level of the reflex centers in the spinal cord (except in the instances to be mentioned later) are the triceps, biceps and radial periosteal reflexes.

The most important abdominal reflexes are the upper and lower abdominal and, in the male, the cremasteric reflexes. These may become exaggerated with pyramidal tract disease, or may be diminished or absent under conditions which will be spoken of in the next chapter.

In the lower extremities compression or a lesion of the pyramidal tracts will cause an exaggeration of the patellar or quadriceps tendon and the Achilles tendon reflexes. As explained in a preceding chapter the pyramidal fibers have a kind of inhibitory effect on muscle tone and hence upon tendon reflexes, and fibers from the posterior nerve roots and also fibers in the cerebellospinal tracts exert a stimulating influence upon the cells in the anterior horns and hence upon muscle tone.

Therefore the patellar reflexes (and in a similar manner other tendon reflexes) are increased by irritative conditions of the posterior spinal roots, disease in the pyramidal tracts by diffuse affections of the spinal cord. The knee jerks are diminished or disappear in disease of the anterior spinal roots, in complete destruction of the posterior roots and posterior columns, in disease of the gray matter at the level of the cells which form part of the reflex arc, in complete destruction of the cord above this level and in deep coma and deep anesthesia.

Marked increase of any one of these may result in clonic contractions. In general Achilles tendon or ankle clonus is most frequent. Lesions of the tracts under discussion may also cause the appearance of pathological reflexes, the Babinski phenomenon (dorsal flexion of great toe when sole of foot is stroked); "Mendel-Bechterew" (plantar flexion of second to fifth toes, frequently with separation of toes, when the region of the fifth metatarsal bone is tapped); "Chaddock" (dorsal flexion of large toe upon irritation of the inner or outer side of foot below the inner or outer malleolus); "Oppenheim" (dorsal flexion of the large toe when the muscles of the calf of the leg are grasped with the hand and massaged in a downward direction); "Gordon" (when the muscles of the calf are suddenly squeezed with the fingers of one hand), etc.

The extensor reflex of the large toe, named "Babinski," after its discoverer, is most often present in spinal disease; the "Mendel-Bechterew" is very frequent, especially when there is marked spasticity.

The tibial periosteal reflex, which consists of a contraction of the adductors of the thigh when the inner surface of the tibia is tapped with a percussion hammer, is regularly exaggerated with pyramidal tract lesions. Sometimes the adductors of the opposite (affected) limb will contract when the patellar tendon of the normal side is tapped (contralateral adductor reflex). This reflex is due to irritation of the uncrossed pyramidal fibers and is very often to be obtained. The *Réflex de défense*, to which Babinski has recently again called attention, is an expression of the automatism of the spinal cord when freed from the influence of higher centers. Defensive movements due to automatic spinal activity have been long known to physiologists. It remained for Babinski to demonstrate their value in spinal localization. In paraplegia from compression of the spinal cord, for example, when a systematic examination of these defensive reflexes from the lower extremities upward is made, part of the cord beyond which no *réflexe de défense* occurs corresponds to the lower limit of the compression. These reflexes (*e. g.*, dorsal flexion of foot, flexion at knee and at hip when plantar surface of foot of paralyzed lower extremity is irritated, flexion at wrist and elbow on irritation of dorsum or plantar surface of hand, etc.) are especially present in level lesions such as transverse myelitis or compression paraplegia, due to fracture of the spine, extramedullary tumors, etc. The contralateral plantar reflex (slow dorsal flexion of the toe of the paralyzed leg on irritation of the sole of the healthy foot) is sometimes observed, when all other signs of pyramidal tract lesion are wanting. I have twice observed this crossed toe reflex among the early signs of spinal compression.

3. **Bladder and rectal disturbances** are very frequent in diseases of the spinal cord. The gray matter of the sacral cord and conus terminalis contains centers for the bladder, rectum, and sexual apparatus. The control of the bladder is due to the tonic contraction of the vesical sphincter. If the bladder is distended, the sensory nerves are irritated and stimuli are carried to the spinal centers and through the third and fourth anterior sacral roots to the bladder muscle. There is an antagonism between the sphincter and the detrusor of the bladder, and it is probable that stimulation of the sphincter is regularly associated with an inhibition of the detrusor, although both are controlled by centers in the brain and by volition.

Disturbances of the bladder may therefore result from disease of any part of the spinal cord. If the disease is located above the spinal centers for the bladder, the control of higher centers is cut off, the patient may lose volitional control, the bladder empties itself reflexly as soon as it is distended, or it becomes overdistended and overflow occurs. If the centers in the cord are destroyed, all control is lost and there is incontinence of urine. The rectum is controlled by a similar nervous mechanism. The earliest bladder and rectal symptoms of spinal disease are generally retention or difficulty in starting the expulsion of the urine, and constipation. Complete incontinence of urine may occur without rectal disturbance, but constipation is more frequent. Finally, incontinence of urine and of feces occurs as a regular symptom of advanced lesion of the spinal cord at any level. Disturbances of the sexual organs are frequently associated with bladder and rectal disturbances.

PART 5.

THE SYMPTOMS OF SPINAL DISEASE AT DIFFERENT LEVELS AND IN DIFFERENT REGIONS OF THE CORD.

Because of the existence, at different levels of the spinal cord, of centers for particular and peculiar functions—the control of the dilator of the pupil, of the bladder, the rectum, the sexual function, etc.—it follows that disease in different segments of the cord will be characterized not only by motor and sensory symptoms from interference with cells in the gray matter and with ascending and descending fiber tracts, but also by disturbances in these peculiar functions.

Thus disease in the cervical region will cause pupillary symptoms, and hence such pupillary disturbances have great importance for spinal localization. If the spinal disease exists in those segments of the cord in which lie the reflex centers for the cutaneous abdominal reflexes, these will be lost, and this loss of one or other abdominal reflex will indicate the level of the disease to which all the other symptoms and signs have to be correlated.

Although a disease in any part of the cord may cause vesical and rectal disturbances, it is especially in conus and cauda equina diseases that early interference with the emptying of the bladder and rectum occurs, and hence early loss of one or both of these functions has diagnostic significance.

Similarly, a disease at the level of the reflex centers for the patellar or the ankle jerks may cause an isolated loss of the one or the other on one or both sides, and the diagnostic importance of such a loss must not remain unrecognized.

The motor and sensory symptoms due to a lesion of the cord vary within wide limits, and depend upon the amount of white and of gray matter affected. In the cervical and lumbar cord, the gray matter occupies a considerable part of the cord, hence in these parts of the cord early muscle atrophies are frequent.

From what has been stated in the preceding chapters, it should be possible for the physician to determine the location of a spinal disease from the symptoms and signs presented. In the present chapter certain peculiarities of symptoms at different levels of the cord will be described.

1. Disease in the upper cervical (from the first to the fifth cervical) segments is sometimes rapidly fatal, but life may be sufficiently prolonged to observe oculopupillary, respiratory, and cardiac symptoms.

The ciliospinal center lies in the gray matter of the eighth cervical and first dorsal segment; it exercises a control over the cervical sympathetic ganglia and is itself controlled by higher centers. When any part of the sympathetic oculopupillary innervation is interfered with, a paralytic miosis (in which the pupil will no longer dilate in the dark and a difference between the two pupils (anisokoria) occurs), a narrowing of the palpebral fissure, and a moderate degree of enophthalmus (sinking back of the eyeball), results. Respiratory disturbances due to interference with the centers for the phrenic nerve and diaphragm (C_3-C_5) has been frequently observed. These respiratory disturbances are frequently caused by a combination of phrenic nerve and medullary lesions. In several instances, however, I have noted that the diaphragm was not paralyzed in spite of extensive disease in the third to fifth cervical segments. It seems to me probable that the diaphragm receives some innervation from still higher centers.

Slowing and irregularity of the pulse is characteristic of high cervical lesions, and is probably due to disturbances in the medulla oblongata, as are also the very high temperatures (103° to 106° F.) that are often observed.

- In these high cervical lesions vasomotor symptoms with disturbances of sweating of the face (Horner's symptom complex) frequently occur. There may be early spastic hemiplegia of an upper and lower extremity with partial loss of sensation on the other side. There may be neuralgic symptoms referable to the occipital minor and major, supraclavicular and auricularis magnus nerves, and

2. **Lesions between the sixth cervical and first dorsal segments.**—In these there is frequently, but not always, a difference between the size of the two pupils (anisokoria) due to a miosis on the side of the lesion, with a diminution or loss of the triceps reflex on one or both sides, marked weakness of the triceps muscle and sensory disturbances, especially on the radial side of one or both upper extremities. The presence of the radius reflex (blow on the styloid process of the radius causing supination of forearm) with absence of triceps reflex is especially characteristic of a lesion at the sixth cervical segment, and I have frequently seen this combination in extramedullary tumors at this level. The sensory and motor symptoms may affect one or both upper extremities, or in addition one or both lower extremities. In the so-called Dejerine-Klumpke paralysis the lesion is at the level of the eighth cervical and first dorsal roots, and the small muscles of the hands and the flexors of the forearm are especially affected. Oculopupillary symptoms are frequent.

3. **Lesions between the eighth and twelfth dorsal segments** are characterized by absence of the abdominal reflexes and by root anesthesias over the abdomen. Sometimes the upper abdominal reflex on one or both sides is absent while the lower is preserved, and vice versa, and by this means it has often been possible for us to more exactly localize the lesion of the cord at the level of the centers for the upper (D8 to D9) or the lower (D10 to D12) abdominal reflexes. The abdominal muscles may not be paralyzed, even though a complete transverse lesion above the eighth dorsal segment exists. I have under my care, at the present time, a patient with a complete crush of the cord at the level of the second dorsal segment, in whom the abdominal muscles are not paralyzed. Hyptonia or paralysis of the abdominal muscles is sometimes a valuable level sign of spinal disease. When the patient coughs, the bulging of the abdominal muscles on either side is very evident. Sometimes, even if there is no paralysis of the abdominal muscles, the difference between the reaction of the muscles to the faradic current on the one or the other side will be very distinct. Girdle pains are most frequent in the middorsal region.

4. **Lesions in the lumbar cord** are characterized by diminution or loss of the patellar tendon reflex on one or both sides if the disease be at the level of or below the second lumbar segment and is frequently associated with early disturbances in the functions of the bladder and rectum. On account of the small size of the lumbar and sacral segments of the cord, disease most often affects a large part of the lumbosacral cord, and it is often difficult to differentiate between the symptoms of this part of the spinal cord and those of the cauda equina. The paralysis is usually of the flaccid type with marked atrophy of muscles. The knee jerks and the cremasteric reflexes are usually lost, while the ankle jerks remain active or are exaggerated, perhaps with ankle clonus.

5. **Lesions of the epiconus.**—Minor has attempted to distinguish the epiconus, in which he includes the fourth and fifth lumbar and the first and second sacral segments. The prominent symptoms of an epiconus lesion are, according to Minor, a degenerative paralysis of the glutei muscles, with especially early loss of power in the peronei muscles. The tibialis anticus is usually not affected; the ankle jerks and plantar reflexes disappear early, while the knee jerks persist. It is very rare, however, that a pure epiconus lesion can be diagnosticated from the signs and symptoms.

6. **Lesions of the conus and cauda equina.**—Clinically we are accustomed to include in the conus the three lower sacral and the coccygeal segments—that part of the cord which lies behind the twelfth dorsal and the first lumbar vertebræ. Disease of this portion of the cord can frequently not be distinguished from an affection of the cauda

equina. Disease of the conus is characterized by retention or incontinence of urine, constipation or incontinence of feces, impotence, anesthesia over the scrotum and around the anus and genitals, without any motor or reflex disturbances in the lower extremities.

In disease of the cauda equina, on the other hand, pain in the small of the back, extending into the perineum, the genitals, and often down the posterior surfaces of the thighs into the areas of distribution of the peroneal nerves, is far more frequent. The sensory loss is often asymmetrical, extending down the posterior surfaces of the thighs and outer aspects of the legs.

The lower the disease of the nerves of the cauda equina the smaller the number of roots that are affected and the smaller is the area of sensory disturbance, but there are diseases which affect both conus and nerves of the cauda equina (soft newgrowths) in which there is very little sensory loss. The characteristic sensory loss in conus and cauda affections varies within wide limits; sometimes characteristic segment loss occurs, at other times the sensory disturbances are very irregular. In both conus and cauda equina lesions weakness of dorsal flexion of one or both feet with more or less complete dropped foot occurs. This drop foot may be the only evidence of a motor disturbance in the lower limbs.

The characteristic symptoms of conus and cauda lesions are pain in the back, rectal and vesical disturbances, diminution or loss of sensation over the areas of distribution of the sacral nerves, loss of ankle jerks and dropped foot on one or both sides with sensory disturbances in the lower lumbar and sacral root areas, flaccid paralyses and atrophy of the muscles of the posterior surface of the thigh, of the leg, and the foot.

The anesthesia extends over the mucous membrane of the rectum, bladder, scrotum, and penis (vulva in women), around the anus, over root areas down the back of the thigh, etc.

THE BROWN-SÉQUARD SYNDROME.

The most frequent combination of symptoms in spinal-cord disease is the Brown-Séquard syndrome, in which motor symptoms occur on the same side of the body below the level of the lesion, and sensory disturbances on the other side. The Brown-Séquard symptom complex occurs where one-half of the diameter of the cord is affected, and in its typical form presents the following:

I. On the same side as the lesion:

(a) Motor paralysis or paresis, due to the interference with the pyramidal tracts.

(b) Vasomotor disturbances due to interference with the homolateral vasoconstrictor fibers in the lateral columns.

(c) Disturbances in deep muscle sense (bathyanesthesia of Oppenheim), with ataxia due to interference with the posterior columns and spinocerebellar tracts.

(d) Superficial cutaneous hyperesthesia.

II. On the opposite side:

Disturbances of sensibility, especially those for pain and temperature (often also tactile).

In addition, on the side of the lesion there are girdle pains and hyper- or anesthesia due to the nerve roots that are involved, so that (on the side of the lesion) the sensory disturbance is on a somewhat higher level than on the opposite side.

If the lesion involves one-half of the cord in the lumbosacral regions, the motor and the sensory loss are usually on the same side, because at this low level few sensory fibers have already crossed to the other side.

The Brown-Séquard syndrome is most frequently observed after injury of the cord due to fractures of the vertebræ or to bullet and stab wounds. It also occurs in spinal tumors and spinal syphilis, and (rarely) in myelitis and in multiple sclerosis. The typical Brown-Séquard symptoms are, however, rarely observed; there is usually a preponderance of motor symptoms on one and sensory symptoms on the other side as an indication of the Brown-Séquard symptom complex.

In some cases of spinal tumor I have observed a reverse condition, motor symptoms on the contralateral and sensory symptoms on the homolateral side. The explanation for this will be given in the chapter on spinal tumors.

Dissociation of the syringomyelitic character is often observed with spinal symptoms of the Brown-Séquard type; on the side of the sensory symptoms tactile sensation is normal or only slightly disturbed, while the pain and temperature sense is markedly affected or completely lost.

PART 6.

THE OPERATION OF LAMINECTOMY.

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Anesthesia.—In most instances the operation is carried out under general anesthesia, although occasions have arisen when I have thought a general anesthetic might be undesirable, and under such circumstances, referred to below, I have been able to complete the operation under local anesthesia. With these exceptions ether is used in my clinic and administered by the endotracheal insufflation method of Meltzer. The apparatus designed by my associate, Dr. George P. Müller, has proven in every respect satisfactory; it is less complicated and less expensive than many others on the market. The tube is introduced with the Jackson laryngoscope, and after the patient has been placed on the operating table no concern need be given to the patient's position, except as above noted, other than to avoid pressure of the neck upon the upper edge of the table: Endotracheal insufflation anesthesia has very peculiar advantages in spinal operations. As the term implies, anesthesia is maintained by ventilation of the lungs with ether vapor under a regulated pressure. The ventilation of the lungs may be effectively accomplished entirely without the assistance of the act of respiration, and indeed oxygenation of the blood is adequately maintained. Whether respiration be embarrassed by paralysis of the intercostal or abdominal muscles, as it sometimes is in spinal lesions, or by the prone position of the patient on the table, the insufflation of the lungs with air, charged with ether, by the endotracheal apparatus is quite competent not only to maintain an even anesthesia, but to oxygenate the blood adequately throughout the operation. For these reasons the use of the apparatus is strongly to be recommended not only as a matter of convenience but as a means of increasing the margin of safety.

Local anesthesia.—Under certain conditions, notably when the patient's vitality is greatly depreciated by the disease and when, as in trauma of the lower cervical cord with paralysis of the intercostal and abdominal muscles, there is the greater risk of pneumonia, I have resorted to local anesthesia. The skin and musculo-aponeurotic layers are readily rendered anesthetic by the infiltration of 0.5 per cent novocain, and while it might be possible to

anesthetize the periosteum of spines and laminae, though not so effectively, when the time comes to remove these, I resort to nitrous oxid-oxygen anesthesia. Novocain infiltration of the cutaneous and muscular layers with nitrous oxid-oxygen anesthesia for the skeletal work has proven a very happy combination and may be considered an important factor in minimizing risks in desperate cases. The patient is given hypodermatically morphine sulphate 0.01 and atropine sulphate 0.0016 one-half hour before the operation; the skin along the projected incision is infiltrated with 0.5 per cent novocain solution, and after the skin flaps are reflected on either side, the aponeurosis and the muscles down to the spines and laminae are infiltrated with massive doses of the same solution. For one operation 250 c. c. may be required. When the spines and laminae are exposed, nitrous oxid-oxygen is given and continued until the bone work is completed. The spinal dura is more sensitive than the cranial dura and before it be incised, either it must be injected with novocain or the nitrous oxid-oxygen continued.

While this method has proven satisfactory in a number of cases, I have been impressed with the greater effectiveness of regional anesthesia, especially in operations upon the thoracic region. The sensory supply of the structures in the field of operation is derived from the cutaneous rami of the posterior primary divisions of the spinal nerves. Following the technic for alcoholic injection of the intercostal nerves, as many nerves on either side as may be necessary, according to the number of laminae to be removed, are injected with 0.5 per cent novocain solution. It may be necessary to supplement these primary injections with injections directly in the line of incision and subperiosteally on either side of the laminae. By a combination of these injections, I have been able to open the spinal canal for various lesions without the aid of a general anesthetic.

One or two stage operation.—The question of a one or two stage operation is often the subject of discussion, but I do not believe in an attempt to settle it, either pro or con, in an arbitrary manner. Each case is a law unto itself, but the decision rests with the judgment of the operator. I depend almost altogether upon the blood pressure as the most valuable index to the patient's condition. If the blood pressure be maintained at an approximately normal line in the preliminary stage of exposure, there is no reason whatsoever for discontinuing the operation until a second sitting. And the more attention one pays to the control of hemorrhage and the avoidance of unnecessary trauma, the less frequently will the operator be forced, as a measure of safety, to postpone the completion of the operation until a second sitting. As a matter of fact, I have had to resort to the two-stage operation but twice. The pulse rate and blood pressure are recorded

on a special chart at five-minute intervals by an assistant, and a glance at the chart, constantly within view of the operator, serves to keep him accurately and continuously informed as to his patient's condition.

Instruments.—The mechanics of removing the spines and laminae are comparatively simple, and few instruments are required. Each operator may determine for himself the instruments with which he can remove the spines and laminae with greatest facility and with the least expenditure of time or infliction of damage. While a great variety of instruments has been suggested and recommended, I have found a modification of the Liston forceps for the removal of the spinous processes and rongeur forceps of various sizes and angles for the removal of the laminae meet every requirement. As with all operations upon the skeleton, the bone-cutting instruments should be sharpened before every operation. Some surgeons employ saws for the laminae, such as the Gigli saw, Horsley's saw, or Doyen's circular saw, others again the conical trephine, and while these may prove satisfactory in the hands of those who use them, the selection of one instrument or another must be considered as matters of personal preference.

In addition to the bone-cutting instruments, but few instruments appropriate especially for spinal operations are required in the armamentarium. Among these may be mentioned two pairs of self-retaining retractors with curved teeth which are particularly helpful in that they maintain an even and constant retraction of the muscles throughout the operation and, at the same time, release an assistant's hands for other duties. Horsley's wax is required to control bleeding from the cut sections of laminae. Mosquito forceps may be required after the dura is opened, and for cutting the theca or roots a pair of iris scissors. For the separation or isolation of roots and sometimes for retraction of the cord I have used hooks of glass instead of metal; these serve a twofold purpose. Being fragile, they serve as a constant reminder to operator or assistant of the obligation to employ the minimum amount of traction force, and as they are nonconductors, they do not transmit to an adjacent root or to the cord the electric current that we may be using for identification of individual roots. In the closure of the wound several kinds of needles should be available: For the dura, a small cutting edge, curved iris needle; for the splint sutures, a full curved Hagedorn needle; for the muscles or fascia, an Emmet needle; and for the skin a straight Hagedorn needle. The suture material includes arterial silk, iodine catgut, and silkworm gut.

Localization.—In preparation for all intraspinal operations, excluding those for trauma, but more particularly for resection of the roots, some means should be adopted for accurate localization of at least

one spinous process. This is best accomplished by selecting for identification the spinous process about the middle of the contemplated laminectomy. Using the seventh cervical or vertebra prominens as a starting point in the cervicothoracic region, or the last thoracic in the thoracolumbar region, the successive spines are counted until the desired one is reached and marked with an aniline pencil. A strip of metal is fastened to the skin at this level and a röntgenogram made. The latter will determine whether the first calculation be correct.

The operation.—With this preliminary operation, with the patient's position properly adjusted, skin disinfected, and the field of operation surrounded with sterile sheets, the operation begins. The incision is semilunar in shape, so that, upon reflection of the flap, adequate exposure is afforded of the structures beneath. The incision proportionate to the predetermined number of laminae to be removed should be long enough to include at least one spinous process above and below the contemplated opening, since it so often happens that one additional lamina above or below may have to be removed. This initial incision extends to the level of the intervertebral fascia. The margins of the flap, including the skin and subcutaneous tissue, are then covered with cloths as a means of protecting the deeper structures from skin contamination.

The incision in the intervertebral fascia begins in the median line at the tip of the spine above the first spine to be removed, and, following closely the lateral aspects of the spines, first on one then on the other side, terminates in the median line just below the last spine to be included in the laminectomy. The incision in the intervertebral fascia is made with a heavy knife, and should penetrate the muscular sheath. The separation of the muscular layer from either side of the spines and from the laminae may be done rapidly with a broad chisel. The preservation of the periosteal layer, partially at least, makes possible more or less bone regeneration, so that the defect consequent upon the removal of the spines may be in part repaired. As a matter of fact, complete bony arches, replacing the original laminae, have been found at secondary laminectomies.

At this stage of the operation there will be more or less bleeding; more if the chisel penetrates the muscles, less if it hug closely to the spines, but hemorrhage is readily controlled by tamponing the wound at once with compresses wrung out in hot normal salt solution. Upon separation of the muscle layer on one side the wound is tamponed before proceeding with the other side, or even before the muscles have been separated from the laminae below. Thus, by working first on one side then on the other, complete separation of the muscles from spines and laminae is effected with a minimum amount of bleeding, and by the time this has been accomplished and the tampons

removed, hemorrhage is well under control. It may be necessary to ligate several bleeding points in the belly of the muscle or in the sheaths and aponeurosis, at which level the largest blood vessels will be found. Intramuscular injections of adrenalin solution, as recommended by some, are unnecessary.

The self-retaining retractors are now introduced, one or two pairs, according to the length of the wound, and beneath them, covering the exposed muscle surface, are spread gauze pads. The blades of the retractors are separated sufficiently to bring into view the articular processes on either side. Having divided with a heavy bladed knife the ligamenta interspinalis above and below, the several spinous processes are removed one by one with bone-cutting pliers and the corresponding laminae with rongeur forceps. In removal of the laminae an opening is made with a small pair of forceps, or, as in the lumbar region, with a drill, and as soon as the space is large enough the heavy forceps are substituted, and the several arches to either side as far as the articular processes, if necessary for proper exposure, are hastily removed piecemeal. While the cord is protected throughout the spinal canal by the epidural fat and in the lumbar region by the ligamenta subflava, care must be taken to avoid injuring the cord in the removal of the laminae. This is especially true in cases of marked kyphosis, where the dura is in closer contact with the arches, and in fracture-dislocations, where the cord may be compressed by the displaced vertebrae.

Once the laminectomy is completed and before the dura is opened, the operator surveys the field in search of bleeding points. Hemorrhage from every source should be under control before entering the dural sac, and to this end the operator will avail himself of one means or another, according to the source of bleeding. If from the bone, sterile wax is the most effective, and if from other structures, heat in the form of tampons wrung out in hot saline solution, tampons of cotton or small pieces of muscle tissue will usually meet every contingency.

The subsequent steps of the operation will depend upon whether the lesion be extradural or intradural. Before opening the dura at least a careful inspection should be made for extradural processes, possibly a tumor, a deformity following fracture, tuberculous foci or what not, and finding the lesion extradural, further exposure is obviated. It would be especially undesirable to invade the subarachnoid space and expose the spinal membranes or cord to infection, should the process prove to be tuberculous. A fusiform swelling of the dural sac, if present, suggests an intradural lesion and gives a clue as to its location.

Before the dura is exposed to view the thin layer of fat, together with the plexus of veins, must be displaced to either side. A careful

inspection of the dura at this stage may bring to light the nature of the lesion. Under normal circumstances the dura is of a bluish-white color and of about the thickness of the dural covering of the brain. Discoloration of the dura with or without the presence of granulation tissue bespeaks an inflammatory process. The absence of pulsation is, of course, most suggestive of a tumor, although both the arrest of pulsation and the oval swelling may be caused by a circumscribed pachymeningitis. To open the dura introduce two silk traction sutures, one on either side, mounted on small curved needles, which enter but do not penetrate. Slight traction upon these sutures frees the dura, and between these a minute incision is made with a small scalpel down to but not including the arachnoid. The latter lies in such close apposition to the dura that in some cases even with care the scalpel will puncture it and the cerebrospinal fluid gush forth. But if not perforated, the arachnoid will balloon through the dural incision under the pressure of the pent-up fluid. The dural incision is lengthened by cutting over a director first in one direction, then in another. Before incising the arachnoid, four additional traction sutures are introduced through the margin of the dura—two above and two below those already in place. The three pairs of traction sutures are grasped separately with mosquito forceps, which by their weight alone will retract the dural flaps sufficiently to afford ample inspection of the subarachnoid space. But before retracting the flaps a roll of cotton a centimeter in diameter and the length of the dural incision is laid in the bed of the wound to the outer side of each dural flap. These cotton rolls will absorb the blood that may gravitate to the bottom of the wound during the operation and prevent its entering the subarachnoid space. If at any time they become saturated fresh ones should be substituted. While perfect hemostasis is unquestionably a *desideratum*, in the strictest interpretation this is not feasible, and yet it is entirely possible to prevent leakage into the subarachnoid space. For this reason the cotton roll plays a very important rôle.

The operator pauses now to inspect the field, and with the arachnoid still intact, his observations may give a clew to the seat and character of the lesion. Under normal circumstances the cerebrospinal fluid pulsates synchronously with the pulse and with respiration. In many instances the respiratory movements may be seen when the pulsations are not visible. This natural phenomenon may not be present if the subarachnoid space above the opening be so obstructed that free communication with the subarachnoid space above is interrupted. In addition to the presence or absence of pulsation, the operator informs himself as to the degree of tension of the cerebrospinal fluid, and as to whether the cerebrospinal fluid be excessive; he notes whether the excess of fluid be evenly distributed

or circumscribed as in circumscribed serous meningitis. In the case of intradural tumor an excess of fluid is found usually above the tumor, though occasionally below, but in either case the fluid may be under such tension that not the slightest pulsation can be detected. Once the subarachnoid space is open the cerebrospinal fluid escapes under varying degrees of tension. Through the initial puncture of the arachnoid I have seen the fluid spurt into the operator's face.

The importance of hemostasis and its means of accomplishment have been discussed, so that it suffices at this time to call attention to them merely by way of emphasis. The second important injunction, the avoidance of trauma, applies, of course, only to the steps of the operation that have to do with the spinal membranes, the roots, and the cord.

I have been convinced of the evil effects of trauma by many operative experiences, particularly those upon the roots. Not until it becomes necessary to manipulate the roots, as in separating the anterior from the posterior, will any material change be noted on the pulse or blood pressure. The application of a pledget of cotton, saturated with 1 c. c. of 0.4 per cent stovain, to the cord and roots at the site of manipulation or just above it has a positive inhibitive influence.

While I do not believe the sudden escape of cerebrospinal fluid is the cause of spinal shock, I can readily see how undesirable it would be to allow the dural sac, ventricles, and cisterns to be emptied of their fluid contents during operation. The continuous flow of fluid is prevented partly by arching the patient's back so that the opening in the dura is at the highest level of the subarachnoid space, but to keep the field entirely dry, posture must be supplemented with small tampons of cotton gently introduced between cord and dura just above and below the upper and lower limits of the dural incision. With this preparation the operator is prepared to inspect the meninges, roots, and cord, and the subsequent steps of the operation will depend upon the nature of the lesion.

Closure of the wound.—Before closing the dural wound hemorrhage must be under absolute control. The presence of even a small amount of blood with its fibrin content leads inevitably to the development of adhesions, and though this may not lead to serious consequences, every effort should be made to avoid them and to leave the structures within the sac as nearly free from the effects of trauma as possible. But a freer hemorrhage within the sac is responsible unquestionably for some of the functional disturbances, either transitory or permanent, that have followed laminectomies. The presence of complete or partial paralysis of one or both extremities, or of bladder or rectum, may be the result of trauma incidental to traction upon the cord or roots or possibly to the resulting edema, following

exploratory laminectomies, but the persistence of these motor phenomena should be charged in most instances to hemorrhage. Perfect hemostasis is, therefore, a *sine qua non* in preparation for closure of the dura. There are occasions when with propriety the dural incision may be left unsutured. Pachymeningitis hypertrophica cervicalis chronica and inoperable tumors are conditions in which for the relief of pressure it is advisable not to suture the dura, and in meningitis serosa chronica there are some who believe in the advantages of providing for drainage, thus preventing the reaccumulation of cerebrospinal fluid. With this view, however, I am not in accord.

To close the dural incision a small, curved iris needle and 00 silk should be used. A continuous suture is preferable to an interrupted suture as a safeguard against leakage and the establishment of a cerebrospinal fistula. The needle is introduced precisely in the edge of the dural incision, the suture is continuous, and with the wound thus closed, no foreign material will be exposed within the dural sac and there can be no leakage through the needle punctures and no leakage along the line of suture. Observing these details in the technic of dural suture, I have never seen any escape of cerebrospinal fluid after laminectomies.

To obliterate the "dead space" that remains after removal of the spinous processes and laminae, three or four splint sutures, according to the length of the incision, are introduced, and after closure of all the layers of the wound is completed, one end of the suture is threaded through a section of narrow rubber tubing so that when tied the suture will not cut through the skin. The next row of sutures of continuous iodine catgut brings the sheath and margins of the erector spinales into apposition, and over this the intervertebral fascia is brought into apposition with interrupted sutures of catgut. The intervertebral fascia is a strong unyielding structure and unquestionably plays an important part in preventing such incapacity as might come from the removal of the spines and interspinous ligaments. As giving support to the structures of the back after laminectomies, it plays a part analogous to that of the fascia of the external oblique after operations in the lower abdomen. There remain to be closed, the skin and the superficial fascia, which are brought into apposition separately, the latter, with interrupted catgut sutures and the former with sutures of interrupted silkworm gut. Finally, the splint sutures are tied, the skin again disinfected with iodine and the dressing applied.

After the operation.—The management of the patient after a laminectomy must take into consideration various conditions that do not pertain to operations elsewhere. With decubitus already present, or from the nature of the lesion likely to develop, the patient is placed

upon a water or air mattress. On the bed immediately beneath the dressing I place a square of wax paper covered with sterile cotton to absorb any blood that may ooze through. This is merely a precautionary measure. I have never had occasion to use fixation dressings, either of plaster of Paris or poroplastic felt. There is no indication for fixation, except perhaps in the cervical region, where the use of small sand bags on either side of the head and a strip of adhesive plaster from one to the other, crossing the patient's forehead, meets every need.

When the operation has been in the lumbar region the nurses and attendants should be warned of the risks of contamination of the dressings with the excreta, and as a protection the dressing should be hermetically sealed around the edges as well as over the surface with adhesive plaster. With an incontinent bladder either a suprapubic drain or a permanent catheter is installed, and retained at least until the wound be healed. The wound itself requires little attention; save for the removal of a drain at the end of twenty-four hours, the dressing is not disturbed until the seventh day for the removal of stitches, at which time a protective pad is applied and allowed to remain until no further protection is required.

The escape of cerebrospinal fluid after laminectomies is more or less of a hoax. When the dural wound is closed, as I have directed, with a continuous silk suture properly introduced, no cerebrospinal fluid will escape, and I might even go so far as to say that even when I have had to remove a portion of the dura I have had no apprehension of a cerebrospinal fistula, since the closure of the musculocutaneous wound with four tier sutures, one each in the muscles, aponeurosis, superficial fascia and skin, is an absolute guarantee against leakage. Where cerebrospinal fistulæ have been established there is, of course, the danger of infection, and the convalescence will be embarrassed with intense headache and vomiting, sweating, hyperpyrexia and acceleration of pulse. In some cases it is reported the fistula closed spontaneously, in some the patient died of meningitis. To arrest the flow and to favor spontaneous closure, the foot of the bed should be elevated and, as a guard against infection, the wound should be cared for most scrupulously.

The condition of the patient after the operation in many instances is not such as to give alarm. The pulse may be accelerated, the temperature subnormal, and the skin leaky, but it is unusual to see the patient in a condition even of surgical shock or collapse. In the majority of cases, therefore, with the exception of morphine for the relief of pain, little or no medication is required, and because the sphincters are frequently paralyzed and because the dressing may become soiled by leakage, proctoclysis is not to be employed.

The most constant and often the only subjective disturbance is pain. Whatever may be the experience of others, I find that most of my patients have enough pain to require one or more hypodermics of morphine during the first 24 hours. The degree of pain will depend somewhat upon the nature of the operation, chiefly upon incidental trauma of the roots; so that after rhizotomy or the disentangling of a tumor from the roots, pain will be intense and morphine or codein is given just as liberally and frequently as may be necessary to insure the patient's comfort. The effect of morphine upon the pulse rate under these circumstances is striking, so that morphine is just as clearly indicated as the employment of general cardiac stimulants are uncalled for.

No general rule can be laid down as to the period of enforced rest in bed. The extent of the laminectomy, whether in the cervical, thoracic, or lumbar region, must be taken into account. In a limited resection, as of three or four cervical or thoracic vertebræ, I allow the patient to sit up at the end of the second week, but after more extensive laminectomies, especially in the lower thoracic and thoracolumbar regions, the patient is content to remain in bed two weeks longer. In so many instances the patient is already paralyzed before the operation that the question of his becoming ambulant depends more upon the paralysis than upon how many laminæ were removed.

Complications.—The complications peculiar to laminectomy are chiefly those which arise from intentional or unintentional, avoidable or unavoidable, damage to the cord and roots; that is, motor or sensory disturbance, transitory or permanent. In the removal of tumors, the displacement of the cord, the separation of roots, involves a degree of trauma that may give rise to temporary disturbances of function, more often motor than sensory. Thus, there may be complete or partial paralysis of one or both lower limbs and of bladder and rectum, abdominal distention and anesthesia below the level of the operation. Although distention is a more frequent complication of operation upon the lower thoracic vertebræ, the abdomen becomes distended, sometimes to such a degree as to cause more or less respiratory embarrassment from pressure of the distended bowel upon the diaphragm. When, coupled with this, peristalsis is arrested and the patient is unable to pass gas, the condition presents a picture not unlike that of peritonitis minus tenderness. Fortunately, the distention is of short duration, disappearing gradually in two or three days, and unless the patient be in distress the condition may be allowed to pass untreated. In the more exaggerated forms I have found the use of the rectal tube and 1 c. c. of pituitrin hypodermatically offered the greatest measure of relief.

After laminectomies there may be either retention or incontinence. Retention of urine is really quite common, irrespective of what may

have been the condition of the bladder before the operation, and restoration of bladder function may be a matter of days or weeks. When of short duration, intermittent rather than permanent catheterization is preferable, but if after a few days there is no tendency to immediate restoration of function, permanent catheterization can not be avoided. One can not always rely upon the patient's sensations, as in certain regions, especially the lumbosacral, the patient is unconscious of the sense of overdistention. Incontinence of urine is a very much more grave complication and implies more serious damage to cord or meninges. Whether it will be permanent or transitory may usually be determined by the nature and seat of the lesion. I have seen a number of cases of transitory retention, but only one instance of permanent incontinence.

Paralysis of bladder, rectum, or extremities, developing as complications after operations within the dural sac, wholly irrespective of what might have been due to the lesion, are unquestionably the expression of pressure within or without the cord, and whether they be transitory or permanent depends altogether upon the nature of the cause. It has been said by some that an excessive collection of cerebrospinal fluid may be held responsible for the pressure symptoms. This seems to me possible but unlikely. I believe the most common cause is hemorrhage without the cord into the subarachnoid space or hemorrhage and edema within the cord, and depending upon the extent, but more especially upon the seat, of the hemorrhage, are the symptoms transitory or permanent. The effects of a moderate hemorrhage without the cord into the meshes of the arachnoid would hardly be more than temporary; the same might be said of a transitory edema within the cord. But hemorrhage within the cord may readily lead to permanent structural changes so extensive as to cause a complete transverse lesion. The importance, therefore, of the avoidance of trauma and strict hemostasis is again emphasized.

Pneumonia or hypostatic congestion is a complication of cord lesions rather than of laminectomy, *per se*, but it is mentioned at this time merely to suggest the propriety of elevating the patient's head and trunk as a prophylactic measure. This applies only to cases with paralysis of the respiratory muscles, as in cervicothoracic lesions.

The prevention of adhesions is a pertinent question. If for any reason the necessity of reopening the dural canal arise, the procedure will be devoid of difficulty, providing there are no intramembranous adhesions, and the converse of this may be stated in more forceful terms. The presence of adhesions is a very grievous obstacle to exploration. How to prevent them is, therefore, a vital question. To deny the tendency for them to form is to close one's eyes to the truth, and to account for their existence when reasonable precautions are

taken is somewhat baffling. I have given much time and thought to this problem, and, excluding infection, the possible causes of adhesions to my mind may be narrowed down to hemorrhage and trauma. As for hemorrhage the deposition of the fibrin in the blood between the membranes in the course of the operation provides at once a means whereby a point or area of adhesion may be formed. On this account particularly must minute care be taken to protect the subdural sac from contamination with blood, and to this end bleeding should be controlled wherever possible before the dural sac be opened. But there will be more or less uncontrollable oozing to be provided for, and to protect the sac from this I have suggested the use of tampons of cotton laid one on either side of the dural incision, so that when the dural flaps are reflected with traction sutures, the cotton tampons will prevent the overflow of blood into the dural sac. Thus, it is a comparatively simple matter to prevent the deposition of fibrin within the sac when the source of hemorrhage is extradural. Not so, however, when the hemorrhage is subdural, since the necessities of the situation, the removal of tumors, the cutting of roots, will provoke more or less bleeding, the control of which will tax the resources of the operator.

The second source of adhesions is the exudate that may form in the membranes as a result of trauma or in the process of repair of the dural incision. The endothelial lining of the dura, like that of intima and peritoneum, is so sensitive to the insult of trauma that it demands the greatest respect.

The laminectomized spine.—There has been, and still is, a very prevalent belief—not among surgeons, however—that the removal of spines and laminae robs the patient of the ability to support the weight of the head or body in greater or less measure, and that after laminectomy the strength of the back is seriously impaired. This is an entire misconception of the facts. Necessary as the spines, laminae or intraspinal laminae might seem to be, it is surprising how little the removal of these structures interferes with the carriage of the body at rest and in motion. This general statement must be qualified in two particulars, first, when the structure of the vertebral bodies are tuberculous or the seat of a malignant tumor, the removal of spines and laminae may be a matter of some consequence and call for some methods of artificial support. It has been suggested that under such circumstances the spinous processes, laminae and articular processes above and below the defect may be lashed together with strong silver wire. I have never had occasion to adopt this suggestion, practical as it seems, and would hesitate to do so because of the possible complications of wound repair. Theoretically, the demands for this method of giving greater security to the spine are not very great; if the indication be of malignant disease.

of the vertebral bodies, a spinal brace would answer the purpose quite as well for the time that remains until the patient becomes bedridden, and if the indication be of tuberculous spondylitis, the lesion itself would necessitate the use of a brace or some other fixation appliance. Secondly, the restoration of function in the laminectomized spine will be influenced, not by the number of spines removed but by their location. The only disability that has been brought to my attention was after the removal of the laminae of five lumbar vertebrae. One of my patients, a middle-aged man, suffered more or less discomfort in the form of pain on certain movements and a feeling of lack of support. Naturally there is more strain at the lumbosacral junction than elsewhere, not only because it is at the base of the spine and supports the entire body, but because this is the junction between a movable and a fixed point in the vertebral column, a point which of necessity is subjected to greater strain.

Leaving out of consideration, therefore, laminectomies at the lumbosacral junction and in the presence of softened vertebral bodies, we now have no hesitation in removing as many consecutive spines and laminae as may be required for thorough exploration or for adequate dealing with the lesion itself. The osteoplastic flap is not essential to the maintenance of function, but perfect apposition of the several musculo-aponeurotic layers in the closure of the wound plays a very important part.

PART 7.

ABSTRACTS FROM THE ENGLISH, GERMAN, AND FRENCH LITERATURE ON SPINAL INJURIES IN WAR.

It is difficult to determine the reason, but it is nevertheless a fact, that the war literature of injuries to the spine and cord is noticeably less than that devoted to the skull and brain; so much less indeed, as to be almost scanty. Holmes ventures as an explanation, the fact that a large proportion of the cases of spinal injury die soon after the infliction of the injury on account of shock, plus associated wounds of chest and abdomen. At all events, the literature devoted to spinal injuries is almost scanty.

For this reason then, as well as for the added reason that there is no marked diversity in the opinions of the various authors, it does not seem wise to group the extracts according to nationality.

Fortunately, the subject of spinal injury has been studied most intensively, in particular by Holmes and Collier. The contributions by both these investigators have been so striking that we are impelled to submit unusually full abstracts. A careful mastery of these abstracts, or better still, of the original articles, will furnish all the basis necessary to handle this type of injury in accordance with most modern principles, ill defined and poorly understood as these principles only too often are.

Gordon Holmes: Spinal Injuries of Warfare. *Brit. Med. Jour.*, Vol. 11, 1915. Nov. 27, Dec. 4, Dec. 11.

I. THE PATHOLOGY OF ACUTE SPINAL INJURIES.

The spinal cord may be injured directly by the projectile and either completely or incompletely divided, but more commonly it escapes direct damage by the missile and is injured only by displaced fragments of bone, which either compress or lacerate it; frequently, however, it is not injured directly either by the projectile or by indriven fragments of bone, and the structural changes in such cases can be attributed only to the concussion or commotion effects produced in the cord by a missile which has struck some portion of a vertebra. Spinal concussion is most commonly seen when the projectile has touched either a spinous or transverse process, which it may have fractured or not, but it may be also produced by a bullet which penetrates or perforates the body of a vertebra.

DIRECT INJURIES.

On examining a case in which the spinal cord has been completely divided, we find as a rule a considerable amount of clot and often pieces of bone between its two ends. A few days after the infliction of the wound these are swollen, irregular, and very soft to touch for at least 1 cm. from the point of division; indeed they may be more or less diffuent, and on handling semifluid disintegrated material, frequently stained with blood, which Sir George Makins, from his experiences in the South African War, very accurately described as custard-like, may extrude. After hardening these portions still seem swollen, softened, and oedematous; the outlines and details of their cross section are obscured, and there are often minute or larger hemorrhages within them, chiefly in the gray matter.

Not infrequently the spinal wound is infected, but these appearances are then modified only by the existence of a septic meningitis which may spread rapidly upward and downward from the lesion. It occasionally happens, however, that hemorrhages and early adhesions between the arachnoid and dura, and in the subarachnoid space limit the infection to the wound. Subdural hemorrhages of considerable size also occur, but they are rarely sufficiently large to compress the spinal cord. Hemorrhages, which are, however, generally small and insignificant, are more common in the soft meninges.

Microscopical examination always shows that there are severe and relatively extensive changes in the spinal cord immediately above and below the lesion; for the distance of half a segment at least and often further the tissue is completely softened and none of its normal elements are recognizable.

These secondary changes, which occur in the neighborhood of a laceration or division of the cord, evidently produce further destruction of it. They are obviously degenerative rather than inflammatory, and are due to the edema and circulatory disturbances that occur in the bruised and necrotic tissue on the borders of the injury, spreading into and involving parts which were not directly damaged by the missile. Edema is the most important factor; it seems to affect the vitality and the neuroglial matrix as well as of the nerve cells and fibers, and combined with circulatory disturbances leads to their disintegration.

Distant lesions.—Edema of both gray and white matter with some swelling and softening of the cord is the most constant of these changes. It gradually diminishes away from the wound and often seems to bear no definite relation to its severity.

Hemorrhages of various sizes are often associated with it, but these are less constant; they are generally small punctiform extravasations of blood, which give a mottled appearance to the cross section of the cord, but they are frequently

larger; a large central hemorrhage with a tendency to spread longitudinally in the cord, such as is generally understood by the term "hæmatomyelia," was not present in any of the fifteen cases in which the microscopical examination has been completed. These hemorrhages are found particularly in the gray matter and about the central canal; one of the most common sites is the dorsal horn, where they can obviously interrupt the reception of afferent impulses. In the gray matter they are liable to break up and destroy the tissue, but when small they produce surprisingly little change in the white columns, the extravasated cells merely tracking along the vessels or between the fibers; occasionally, however, there is some local softening, and later neuroglial proliferation, around larger extravasations.

The extent of these small disseminated intraspinal hemorrhages is occasionally surprising; in one case they spread over two and a half segments on each side of the wound. They are found with lesions of all regions of the cord, but they are usually most prominent when the cervical region is wounded and probably least so with injuries of the lower dorsal and lumbar segments. Owing to the relatively slight destruction they produce in the tissues their importance in the production of clinical symptoms may be easily overestimated.

CONTUSION OR COMPRESSION OF THE SPINAL CORD.

When a portion of a vertebra or a detached spicule of bone is driven into the spinal canal it frequently lacerates both the cord and the theca and causes lesions which may differ only in degree from those produced directly by a projectile. Frequently, however, there is no obvious external injury to the cord and the dura mater is not torn, even though the clinical symptoms indicated a complete transverse lesion. Small hemorrhages into the meninges are, however, common, and on palpation the cord at the level of the contusion is soft, and if the pia mater is incised or pricked semifluid custardlike material may escape.

When the injury is less severe the normal appearance of the cross section is only obscured, and there are frequently minute hemorrhages throughout it. The damaged area and the segments on either side of it are usually swollen by edema, and the cord may be indented by the indriven bone. When the lesion is examined under the microscope changes are found very similar to those in the parts adjoining a direct injury, but their intensity naturally varies much. The most important is softening and disintegration of the tissues, always greatest in the region which was directly contused. If the injury is severe the whole cross section may be softened, but more commonly there are discontinuous foci in the ventrolateral and dorsal columns. The gray matter may be also completely destroyed in whole or parts, but it is more usually extremely edematous and only partly disinte-

grated, with its nerve cells necrotic or in advanced chromatolysis. Occasionally only a diffuse or focal necrosis is found in which no stainable elements persist. The affected areas are gradually invaded by granule cells, and a considerable proliferation of neuroglial cells is visible around them, but, apart from a pronounced congestion of the vessels and an occasional increase of the cells in their walls, there is no evidence of any inflammatory process. The amount of hemorrhages into the injured region also varies very much.

The distant lesions differ in no respect from those which are so commonly associated with direct spinal injuries. Diffuse foci of necrosis and softening, vacuolation due to falling out of fibers and the sievelike rarefaction produced by the disappearance of fibers and of the finer glial matrix, as well as minute scattered hemorrhages, are found in the white matter of the adjoining segments, while the gray matter is also edematous, and contains similar hemorrhages and perhaps foci of softening. The central cylindrical cavities, which have been already described, also occurred in the dorsal columns relatively as frequently as with direct injuries.

CONCUSSION OF THE SPINAL CORD.

In cases of concussion, when the cord is not damaged by the fracture or dislocation of a vertebra, there may be no external signs of injury, or only a more or less uniform swelling opposite the site of impact, and, even to touch, no definite abnormality may be recognizable.

On microscopical examination the vessels are found engorged, and there are generally punctiform hemorrhages, especially in the gray matter. The most striking change, however, is the oedematous swelling of the most affected segments with either diffuse or focal necrosis and softening, which, at least in the cases that have been examined microscopically, has been most pronounced in that part of the cross section nearest the point of impact. In these areas there may be complete destruction of all the functional elements, but more usually only a proportion of the fibers have disappeared, while the myeline sheaths and axis cylinders of others are swollen. Focal softenings also occur in the gray matter, but are usually unrelated to the hemorrhages which this frequently contains. There is, often, however, some softening and disintegration of the tissue, as well as degeneration of the nerve cells, around these hemorrhages.

The distant lesions in cases of concussion are similar to those found associated with direct and contusion injuries, but they are often very marked in relation to the changes found at the site of maximum damage. Scattered hemorrhages, irregular foci of necrosis, and softening and cavity formation occur, but the most pronounced feature is the extensive parenchymatous changes that often extend over four or five segments in either direction. These consist in the

swelling of fibers, either isolated or in groups, in the midst of tissue which is otherwise normal or only slightly cedematous; as a rule the axis cylinder is more swollen and the myeline sheath surrounds it as a distended and attenuated ring, but in places the sheaths are more affected, and are often broken up. The disappearance of these swollen fibers and the partial disintegration of the neuroglia leave vacuoles and round or oval cavities in the white matter and give it a reticular or sieve-like appearance. There are also often considerable histological alterations, either degenerative or chromatolytic, of the nerve cells at some distance from the level of the injury, but these are found most commonly where the tissue is cedematous.

We must now consider shortly the nature of these changes which have been described, the causes to which they are due, and their significance in the production of the clinical symptoms that characterize these cases. The conclusions and views expressed must not be regarded as final.

Spinal concussion.—The most important and obscure factor is that which we understand by *concussion*—that is, functional or anatomical disturbances produced indirectly in the spinal cord by a sudden and violent impact on the vertebral column. The nature of spinal concussion has been much discussed, and it has been, in fact, questioned if spinal lesions, such as those described above, do occur apart from temporary dislocation or fracture of a vertebra, compression by fragments of bone, or an extra or subdural hemorrhage causing direct trauma to the cord.

But many cases in which none of these possible causes existed have been recorded, and we have had the opportunity of observing cases in which they could not be demonstrated. Further, certain of the distant lesions that we have already described, which often extend over several segments on either side of the primary injury, or beyond the level of the impact on the vertebral column, can not be due solely to a direct trauma, and they are identical in nature to those attributed to concussion. The changes are irregular focal softenings or patches of necrosis, sieve-like vacuolation of the white matter disseminated hemorrhages and local lesions of the myelinated fibers.

It is in the first place necessary to insist that these changes are not, as oedema may be, continuous with those in the neighborhood of the spinal wound; the hemorrhages, for instance, are usually discrete and are often only minute extravasations of blood in the Virchow-Robin or perivascular spaces, while the focal necroses and softenings do not, as a rule, spread longitudinally in the cord from the region of the trauma. Further, when a group of fibers is affected, the lesion is usually focal, and it is not necessarily those of one tract only that are involved; it can not, consequently, be either a manifestation of a secondary degeneration or of a pathological change which has spread from the point at which the fibers were directly injured.

The special character of these lesions is, therefore, their diffuse and irregular distribution and their tendency to diminish gradually from the point of maximal disturbance. They are not due to hemorrhages, as these bear no constant relation to them, and as vascular occlusions are also rare, these obviously can not be the main causal factor. On the other hand, as the chief lesions are foci of primary necrosis and parenchymatous change in the cells and nerve fibers, the essential changes may be described as primary disturbances in the vitality of certain portions of the tissue, associated with œdema and frequently with small scattered hemorrhages.

It is difficult to offer a complete and satisfactory explanation of how a blow on the vertebral column can produce these lesions in the cord, protected as it is within the canal. Certain structural alterations found in cerebral concussion are attributed to the violent oscillations produced in the cerebrospinal fluid, especially in that of the ventricles, but the spinal cord is only surrounded by fluid, and is able to swing to some extent within the dural sac with its oscillations. The waves of pressure thus set up may, however, produce physical effects within the cord, and possibly disturbance of its lymph circulation, but the most probable explanation is that put forward by Fickler, according to which the cord is made to oscillate within the canal by the impact on the vertebral column, and as its movements will obviously not be synchronous with those of the column it may be directly bruised against the walls of the canal, while at the same time the sudden jarring of the cord produces a physical disturbance in its tissues, and especially in the fluid axoplasm of its fibers. There is much in favor of this explanation, and the factors it hypothecates can not be neglected. The histological changes in the spinal roots may be, in fact, partly due to the strain thrown upon them by the displacement of the cord. But if it is the whole explanation, the structural lesions should be greatest at the position of contre coup; we have, however, so far always found them most pronounced immediately under the site of impact. Further, it must be remembered that the spinal roots and the ligamentum denticulatum limit the movement of the cord within the canal, and in many levels at least must make contre coup bruising impossible.

Whatever may be the exact mechanism of spinal concussion, it must be admitted that a sudden violent impact on the vertebral column can produce diffuse, irregular, and severe structural changes within the spinal cord. The factors which determine the severity of these lesions must be the momentum of the projectile, the part and surface area of the vertebra which it strikes, and the region of the spine which is wounded.

Secondary changes.—Finally we must consider the secondary changes which may occur in the neighborhood of the primary injury and in those portions of the cord which have suffered from concussion.

It has occasionally happened that when the wound is not severe the patient was at first able to perform some movements in his legs, but lost the power to do so within the following two or three days. Further, we have seen new symptoms develop or their level alter under observation. This may be due to a secondary hemorrhage into the affected tissues or to progressive softening, of both of which we found possible evidence in our sections.

But the most striking secondary change was the development of the cylindrical cavities we have described. Their exact pathogenesis is obscure, but certain features they present must be emphasized in attempting an explanation. In the first place, they seem to involve the destruction of very little tissue, but, rather, separate and compress the fibers around them; their contents must be consequently under much pressure. In the second place, they evidently develop away from the lesion, as the material they contain is always less necrotic at their upper or lower end if they are, respectively, ascending or descending cavities; and finally they do not as a rule extend to the maximal lesion, but are generally connected with this by a track of oedematous or softened tissue, or by a narrow channel of softening, or by a fissure. It seems, therefore, probable that they originate from the accumulation under pressure of transuded fluid and degeneration products in a small projection of the primary lesion, which tracks upwards or downwards along the lines of least resistance through either normal or oedematous parts, destroying only a relatively small amount of tissue, but increasing in size probably under the same principles as a retention cyst. The granule cells which frequently line their walls or are contained within them must be due to a reactionary proliferation of the neuroglia in the tissue through which they track.

Finally, it must be emphasized that they are not due to infection, as in several cases in which they occurred the theca had not been lacerated and there was no sign of infection in either the cord or the meninges.

They are obviously not produced by hemorrhages, though a certain number of red blood cells may be found within them, nor by vascular lesions, as there has been no evidence of these, and the position of the cavities does not correspond with the distribution of any spinal vessel. The frequency with which they occupy the ventral portion of one or both dorsal columns is striking, but its significance is not clear. This region is, however, a watershed area between the distribution of the anterior spinal arteries and of the small arteries that supply the dorsal columns, and as such may have a relatively poorer blood supply than other parts of the cord. In many cases, too, the dorsal columns seem to suffer more severely with oedema and softening than the ventrolateral columns.

It is obvious that these irregular and diffuse changes which are found in various types of spinal injury must be taken into account in interpreting and estimating the sig-

nificance of the clinical symptoms that are observed in these cases. The following conclusions at least can be drawn:

1. The structural lesions in the spinal injuries of warfare are rarely sharply limited or circumscribed, and can not be compared to those produced experimentally in a physiological laboratory. The level of the lesion, as indicated by the clinical symptoms, for instance, often does not correspond with the level of maximal damage.

2. The lesions are so irregular in distribution and severity when the spinal injury is not complete that much care is necessary in drawing conclusions from the clinical symptoms alone on the functions of parts which it may be assumed have been involved.

3. Secondary changes may occur later in the cord which can alter or modify the clinical symptoms.

II. THE CLINICAL SYMPTOMS OF GUNSHOT INJURIES OF THE SPINE.

LOCALIZATION OF THE LESION.

The segmental level of the lesion can be usually recognized as accurately by the extent of the motor paralysis as by the upper border of the sensory disturbance; and since the evidence it gives is less equivocal and as easily interpreted in both military and civil practice, some emphasis may be laid on its importance. The segmental innervation of most of the muscles of the upper and lower limbs is now known, and this knowledge has been applied in clinical work. A paralysis of all the movements of the wrist and fingers as well as of the triceps, while the biceps remain strong or only slightly weakened, is usually, for instance, taken as an indication of a lesion in the seventh cervical segment, but hitherto little attention has been given to the evidence of the level afforded by the palsy of the trunk muscles. When, however, one of the lower six dorsal segments is involved, the part and the extent of the muscles of the anterior abdominal wall which are paralyzed form an easy and certain guide to the segment in which the lesion lies. If, for instance, the eleventh is involved the whole rectus abdominis contracts when the patient raises his head, attempts to sit up, or coughs, but the iliac regions bulge owing to paralysis of the lower portion of the obliqui abdominis, and their failure to contract can be easily recognized by the observer's fingers. Similarly, if the ninth segment is injured, it is obvious to the finger that the recti abdominis downwards from about 1 inch above the umbilicus do not contract, but are, in fact, passively stretched by the tension produced on them by the shortening of the upper segments. Owing to the same fact the umbilicus, as Beever first pointed out, rises toward the xiphoid. The state of the intercostals is an equally reliable guide to the level of the injury, and permits a local diagnosis in the upper as well as in the lower dorsal segments. If the fingers are firmly placed in series on the inter-

costal spaces, the unaffected muscles are felt contracting on each deep inspiration, and form a firm shallow floor to the space, while in paralyzed spaces no contraction can be felt, and on deep inspiration the finger sinks deeper between the ribs; in lean subjects this may be, in fact, visible to the eye. As the intercostal muscles have only unisegmental innervation and as each receives its nerve supply from the correspondingly numbered dorsal root, the highest space which is paralyzed indicates the level of the spinal injury.

The upper limit of disturbance of sensation is the means most commonly used in civil practice to determine the segmental level of the spinal lesion, and if proper care is taken the evidence it gives is reliable, but, as we shall see later, in incomplete lesions, and more especially in those which are wholly or chiefly unilateral, errors may easily occur, and an exact local diagnosis may not be always possible from even an accurate sensory chart; this is due to the oblique course of the decussating sensory fibers in the cord. In a complete or very severe lesion, light contacts are usually felt a short distance below the limit of complete analgesia, but there is frequently some disturbance in tactile sensibility above the level of the latter. The appreciation of moderate temperature is often lost slightly higher than that of painful stimuli.

The disturbance in the appreciation of vibration may be also a valuable indication of the level of the injury, especially in incomplete cases in which the dorsal columns only are damaged and sensibility to touch and pain is unaffected, since the vibrations of a heavy tuning fork can not then be recognized below the corresponding segmental area. This can be determined by drawing the base of the vibrating fork upward over the soft parts. This method is particularly valuable on the trunk when the state of the other elements of sensation conducted through the dorsal columns can not be investigated; the base of the fork may be simply drawn over the anterior abdominal wall till the level is reached at which the patient feels the vibrations, but, as the thorax can act as a sounding box and transmit the vibration widely over it, it is necessary to apply the fork here only to folds of skin raised gently between the observer's fingers and thumb.

When one of the lower abdominal segments is involved, the level of the lesion may be also accurately determined by observing the segment below which the abdominal cutaneous reflexes can not be obtained.

It must be remembered, however, that the lesions produced directly or indirectly in the spinal cord by a gunshot wound are often very extensive, and that a clinical examination can, as a rule, indicate only their oral limit.

REFLEXES AND REFLEX TONE.

In all severe lesions the lower limbs are found flaccid at least as early as one day after the infliction of the wound,

and within three or four days their muscles become toneless and flabby; if the lesion is complete or almost so they remain flaccid and waste gradually; later the atrophied muscles, especially those of the calf and the flexors of the toes, undergo fibrous contracture. In less severe cases the muscles regain tone and the limbs become slightly rigid, generally within 14 to 20 days. In one case, however, we observed slight rigidity in a limb five days after the wound was inflicted, but in another spasticity appeared only after 84 days. In slight cases there may be no obvious defect of muscle tone, or, if diminished, as it frequently is at first, it rapidly recovers.

In those cases in which some rigidity develops early reflex spasms of the legs of the flexor type are apt to occur; they are, as a rule, seen only a few days after the limbs have become somewhat spastic, but we have observed them occasionally as early as the sixth to tenth day, when the tone of the muscles was not yet exaggerated.

In one interesting group in which pains due to higher spinal lesions occur in the legs, these limbs are often held stiff and rigid, but careful examination shows that there is no true spasticity, and reflex spasms do not occur; in these cases the spinal lesion is not severe, and voluntary movement is either not lost or has recovered rapidly.

The state of the reflexes in the affected parts presents interesting problems. Except when the spinal lesion is slight the knee and ankle jerks are almost invariably lost at first, and in severer cases remain absent during the period in which we have been able to observe them—that is, in some instances, for as long as 6 to 10 weeks. The teaching of Dr. Charlton Bastian that these reflexes are permanently abolished in total transverse lesions of the cord is generally accepted now, and our experience seems to confirm it, though in one case in which a fragment of shell casing lacerated the cord in the lower part of the fourth dorsal segment and passed downward through the next three lower segments, apparently destroying them completely, feeble knee jerks could be obtained from the fifteenth day onward; whether or not there was a total transverse lesion has not yet been determined by microscopical examination.

In less severe injuries the knee jerks return, but generally not earlier than within two or three weeks; the re-appearance of the ankle jerks is always later than of the knee jerks, but occasionally ankle clonus could be obtained while the knee jerks were still absent or much depressed. In a few cases in which paraplegia in flexion developed after the return of the knee jerks we saw these again disappear as the flexion rigidity increased. In lesions of the upper four cervical segments the arm jerks are usually lost at first, independently of the severity of the injury, and seem to recover less early than the knee jerks.

Not only are both knee and ankle jerks absent for a considerable time in transverse spinal injuries, but with uni-

lateral lesions of the cervical or dorsal segments neither can usually be obtained in the paretic leg for some days, or they are at least much diminished on this side compared with the normal. The paretic leg is also usually flaccid. In these cases, however, the reflexes return earlier than in transverse lesions of the same degree of severity, but usually not till at least 10 to 12 days after the infliction of the wound; in one patient with a unilateral cervical lesion we could elicit only a very feeble reflex after 35 days, and it was almost two months after the injury that the jerks of the homolateral limb were as brisk as normal.

On turning to the superficial reflexes we find the abdominal and cremasteric more easily abolished than the tendon jerks; in fact, when the lesion lies above the mid-dorsal level, both remain permanently absent as long as there is any obvious motor weakness of the lower limbs.

In spinal injuries above the lumbo-sacral enlargement we would expect, on stimulating the sole, to obtain constantly the abnormal type of plantar response originally described by Babinski. When the lesion is complete or particularly severe, however, no movement of the toes may result, and there may be no reflex contraction of the hamstrings or of other muscles; and this holds not merely for the first few days when extensive functional disturbances might be attributed to "shock," but the condition may persist for several weeks at least.

In some cases, however, probably when the transverse lesion is not total, stimulation of the sole produces only a simple flexion of the great toe, often associated with slight flexion and adduction of the smaller toes; this flexion of the great toe can be produced when the outer border of the sole only is stimulated, and consequently it can not be attributed to direct mechanical irritation or stretching of the small flexor muscles of the sole. The movement differs from the normal flexor response in that it is slower and smaller in range, and in that it is chiefly a flexion at the metatarsophalangeal joint. Occasionally the only effect is contraction of the inner hamstrings, but as a rule this is associated with slight flexion of the toes.

In less severe injuries stimulation of the sole still evokes flexion of the great toe with contraction of the hamstrings, while, if the lesion is still less serious, a withdrawal reflex of the whole limb, in which the hamstrings, tensor fasciæ, flexors of the hip, and the dorsiflexors of the ankle are concerned, may be obtained, but still with flexion of the great toe. In many cases, however, an extensor response can be elicited from the sole, but clinical experience and post-mortem examinations tend to show that during the first week or 10 days at least Babinski's sign occurs only with transverse lesions, which are not complete. We have repeatedly seen a flexor movement of the toes give place to an extensor between the seventh and the twentieth day after the injury, and in certain cases this has been a precursor to

improvement. Even in one case in which a unilateral lesion of the fifth cervical segment produced a flaccid paralysis of the limbs of the same side the plantar reflex was absent, or only a slight slow flexion of the great toe could be obtained during the first two weeks, after which it gave way to a pure extensor response.

SPINAL SHOCK.

This state of the reflexes, more especially the abolition of the tendon jerks and the absence of the Babinski's sign, in severe but not necessarily complete anatomical lesions raises many points of interest. Even if we accept Dr. Bastian's doctrine, we must be surprised to find the tendon jerks absent, for a time at least, in such a large proportion of incomplete injuries. This differs from what we find in ordinary civil experience, except in cases of fracture dislocation of the vertebral column, and in this condition the medullary injury resembles that produced by gunshot wounds of the spine. The nature of the lesion can not, however, explain it, and as the reflexes disappear even when the highest spinal segments are injured, their absence can not be attributed to the distant disturbances that have been described in the first lecture. The most obvious common factor is the sudden severance of a portion of the cord from the influence of more orally situated centers by an abrupt section or by a physiological block. This produces the condition which is generally known as "spinal shock." It is recognized in the experimental laboratory, as high transection even in the frog leaves all four limbs flaccid and inactive to stimuli for half an hour or so, and the higher the animal stands in the scale the more pronounced and persistent are the symptoms of shock. In man, in whom the spinal mechanism is most subordinated to the higher centers, the effects of shock are naturally most pronounced, and the caudal portion of the cord is least capable of acting alone as an effective central organ. Our observations, therefore, only extend and confirm the experiences of physiologists, and show that the sudden isolation of a portion of the cord from the rest of the central nervous system leaves it incapable, for a time at least, of subserving even the simplest reflex.

The unilateral absence or depression of the tendon jerks in cases of unilateral lesion is interesting, as it shows that their abolition is not due to a state of general shock or to a sudden gross traumatic injury of the cord, but that it must be attributed to an interruption of impulses that descend through the homolateral half of the cord, which produces a functional depression on this side only.

We have not yet had the opportunity of determining whether, in cases in which the structural lesion is not complete, the absent reflexes eventually return, at what date they reappear, and with what other symptoms of recovery their reappearance is associated. We have, however, seen the knee and ankle jerks absent during the first and second week in

cases which have recovered sufficiently to stand and absent for longer periods in patients who later regained some power of movement while under observation.

The inability to elicit reflex movements from the sole in cases of complete transverse lesions must be also attributed to the functional depression, either temporary or permanent, of the isolated segments of the cord. It has been pointed out that in less severe cases only flexion of the toes or this associated with contraction of the hamstrings is obtained, and that only in less severe or longer standing injuries can the complete flexion reflex be evoked. This we might expect, for when the activities of the isolated portion of the cord are depressed by shock the relatively complex mechanisms of commissural and intersegmental association naturally suffer more than the simpler and more rudimentary unisegmental functions. And as the sole, from which the reflex is most easily evoked, lies within the sensory distribution of the first sacral root, and the flexors of the toes and the hamstrings are innervated chiefly by the ventral root of the same segment, the contraction of these muscles on stimulation of the sole can be regarded as a unisegmental reflex; additional segments would be concerned in flexion of the hip and knee and the contraction of the tensor fasciae femoris and adductors, which are included in the full flexion reflex. Further, in these cases the receptive field of the reflex is much narrowed, and is, in fact, almost invariably limited to the sole, where the threshold of effective stimulation is normally lowest.

It might be expected that the effects of shock on the lumbosacral enlargement would be more pronounced the lower the lesion lay in the cord, but we find little to support this view; the lower limbs have been as flaccid and toneless in cases of high cervical injury as when the lower dorsal segments were damaged, and when the lesions have been probably of equal severity there has been no evidence of less shock or of earlier recovery when it lay high rather than low in the cord. This would support the conclusion drawn from the observation of unilateral lesions that shock is not a direct mechanical disturbance of functional activity, but that its effects depend on the interruption of the neurotic impulses that normally flow continuously from the higher to the lower levels of the central nervous system.

Sherrington has pointed out that the effects of spinal shock are seen in experimental animals only in the aboral direction, and it is obvious that in man they are limited to segments distal to the lesion, as in even the rudest transverse lesion no symptoms are found above its level which are attributable to shock.

"AUTOMATIC" MOVEMENTS.

But, though the shock effect of these severe spinal traumata almost invariably abolishes or depresses seriously the functions of the isolated segments, in a group of four cases

in which the lowest dorsal or highest lumbar segments were involved, "automatic" movements, such as are observed in certain spinal animals, occurred, and their occurrence can be interpreted only as the result of a reflex overactivity of the isolated segments. In one of these cases the lesion involved the first and the upper part of the second lumbar segment; in another it extended from the lower part of the twelfth dorsal to the middle of the second lumbar segment; in a third it reached as high as the eleventh dorsal segment and probably extended some distance downward, while in the fourth it was probably limited to the third lumbar segment. In these cases the lesions were severe or total, and as the involuntary movements were identical or very similar in all four, their nature can be best conveyed by describing one case in detail.

These alternate flexion and extension movements of the lower limbs obviously represent the rudiments of the lower physiological mechanism of gait, and are very similar to the "mark time" or progressive movements seen in the "spinal" dog; their nature must be the same as these reflex movements which Sherrington has described in the spinal animal.

It is interesting, however, that we have seen such involuntary reflex movements only when the lesion involved the upper lumbar segments of the cord, and that they occurred in a considerable proportion of all serious injuries at this level. In the only two cases in which the spinal cord has been examined there was exceptionally little distant disturbance, and the lower lumbar and the sacral segments were in both almost intact. It is also surprising that they should occur at least as early as the second day. Further observations will be necessary to determine the significance of the absence of direct structural damage in the ventral columns in the two cases which came to autopsy.

We have not observed any "automatic" movements of the limbs when the higher cervical segments were injured; in fact, these are always then flaccid and toneless, and their muscles usually wasted early. We have, however, obtained a reflex—which, as far as we know, has not been yet described—in cases in which the lesion lay in or above the fifth cervical segment, and produced paralysis of the upper limbs. Pricking, pinching, or firm stroking on the inner side of the arm then evoked, generally after a short latent period, a sudden and strong inward rotation and adduction of the arm on the same side, the inward rotation being apparently the prime and chief movement. We obtained this reflex in most of the cases of severe cervical lesions in which we examined for it, provided there was not an atrophic palsy of the arm.

CERVICAL SOFTENING.

Injuries in different portions of the cord naturally produce clinical symptoms differing not only in the extent, but

also in nature of the paralysis. One of the most striking types is that which results from an incomplete lesion of the cervical enlargement. Since hemorrhages and secondary changes undoubtedly occur as a result of concussion more readily in this than in any other region of the cord, and since they are more liable to damage the gray than the white matter, the arms are frequently seriously paralyzed, though there is fair or unaffected power of movement in the lower limbs. Not infrequently the paralysis, especially that of the arms, develops some time after the infliction of the wound, but, on the other hand, it is not uncommon to meet patients with gunshot wounds of the neck in whom all four limbs were at first paralyzed, who had regained power within a week or so, the arms recovering almost invariably later than the legs.

Lesions of certain regions of the cord also produce special local symptoms. We have, for instance, repeatedly seen unilateral paralysis of the diaphragm, and in two cases at least bilateral palsy, due to lesions at the level of the fourth or fifth cervical segment; unilateral palsy of the diaphragm also occurred in two cases in which the main lesion was to the second cervical segment, but it was not observed in any patient in whom it lay lower than the fifth. The occurrence of nystagmus has also been described as a result of lesions in the higher cervical region, but we have observed it in only 3 of the 63 cases in which the cervical segments were injured, in lesions of the second, fifth, and seventh segments. It was slight and ill-sustained in all three, and disappeared rapidly, save in one patient, in whom it persisted at least fifteen days; but as in this case the exit wound was in the neighborhood of the tip of the mastoid labyrinthine concussion can not be here excluded.

PALSY OF THE CERVICAL SYMPATHETIC.

Disturbances of the functions of the cervical sympathetic occurred with lesions of all segments between the second cervical and the second dorsal included; they are referred to in our notes on 36 cases, and in the great majority, at least, of these injury of the sympathetic fibers in the neck could be excluded. The most common and prominent symptom was miosis, or, in unilateral lesions, inequality of the pupils, the smaller being on the side of the lesion, and this, as a rule, failed to dilate, or dilated less rapidly and less completely on shading the eyes. A narrowing of the palpebral fissures in one or both eyes and some enophthalmos was also pronounced in most of the cases. Ptosis was also frequently observed, especially with lesions of the lower cervical and the first dorsal segments.

Disturbances in sweating on the affected side were also present in most of the cases; as a rule, the skin of the face, neck, and shoulders, as well as the hair of the head on the side of the spinal injury, was merely drier and less

greasy than on the opposite side; but during the warm weather of the late summer, or in any condition that induced sweating, there was a very obvious difference, for the skin of the face, neck, and shoulder to the level of the second rib remained dry on the one side, while it was moist and covered with beads of sweat on the other. In certain unilateral lesions, too, a diminution of tear secretion was observed on the affected side, the eye being obviously drier and presenting a more staring and glassy appearance than the normal; a few patients even complained of this eye being "stuck" or difficult to open in the morning, owing to the lids being adherent as a result of the drying of the undiluted conjunctival secretion.

Definite vasomotor disturbances associated with paralysis of the cervical sympathetic were less common, but in several cases the face was more flushed and highly colored on the affected side, especially after shaving.

It is known that the cilio-spinal center lies in the lowest cervical and first dorsal segments, while the spinal center of the other components of the cervical sympathetic is found in the two upper dorsal segments, and that these are influenced or controlled by efferent bulbar fibers which descend uncrossed through the cervical cord. It must be to disturbance of the latter that the symptoms just described are due when the lesion lies above the eighth cervical segment, while the spinal centers themselves are injured when the lesion lies below this. It is an interesting question if the symptoms due to lesions in those two sites differ. We have not, however, observed any essential or obvious difference, though both the ocular and secretory disturbances seemed to be on the whole more prominent and permanent when the spinal sympathetic centers were damaged than when the bulbar efferent fibers were involved; in fact, in the latter case the symptoms usually subsided quickly, and often disappeared under observation. Symptoms of irritation of the cervical sympathetic did not occur in any case in which the spinal cord only was injured.

HYPOTHERMIA.

One of the most interesting types we have observed was due to injury of the lower part of the cervical enlargement, and was characterized by subnormal temperature, slow pulse, low blood pressure, and scanty secretion of urine. All the 10 patients in whom these symptoms occurred died within eight days after the infliction of the wound, and in all the lesion lay in approximately the same region.

On admission to a base hospital, generally one or two days after the injury, the most striking feature was the cold, collapsed condition of the patient. The skin was generally dry and remarkably cold even to touch, and it was noted in some that the superficial temperature on exposed parts, as the face or hands, was apparently no lower than that of covered parts. When the temperature was very low, a touch re-

mind one forcibly of the coldness of death—this was especially so in a man seen at a casualty clearing station soon after he received the wound. The skin was also generally cyanosed, and the face of a curious slate color. The temperature varied in different cases, and as unfortunately a special thermometer to register it was not always available, in 4 of the 10 cases we can only say that it was lower than could be registered in a clinical thermometer—that is, $36^{\circ}\text{C.}=95^{\circ}\text{F.}$ In one of the other six it sank to 78.8°F. (26°C.) in the rectum, and did not rise above 80°F. during the 24 hours he lived under observation, but in most of those in which accurate observations were made the lowest recorded lay between 80° and 84°F. In a few it rose suddenly before death to above the normal, and in one case which has been already recorded by Lieuts. Oliver and Winfield actually rose from 80° to 105.6°F. —that is, 25°F. —in 26 hours; in others it remained low till the patient ceased to live. In one case the patient lived at least three days with a temperature not rising above 90°F. , and in two it did not exceed 85°F. for 24 hours, but as these cases usually did not come under our observation till the third day, it is probable that life could be maintained even longer with this low temperature.

The temperature was always taken in both the mouth and axilla, and in a few instances in the rectum, too; the thermometer in the mouth generally registered slightly higher than in the axilla, but, except in one case in which it was a few degrees higher, the rectal temperature was approximately equal to that in the mouth.

In all cases, too, the pulse rate was very slow, while the temperature remained low, and it increased in frequency as this rose; in one case it was only 22 per minute, and in the others ranged between 30 and 50 per minute till the temperature approached normal limits. In one patient it was 32 when the temperature in the mouth was 80.6°F. , and rose to 102 when this reached 98.8°F. The pulse was also very soft and of low tension while the temperature remained down; unfortunately, sphygmomanometric observations were possible in only three cases, and in these the pressure registered 56, 72, and 73 mm. of mercury.

As the intercostal muscles were paralyzed in every case, respiration was wholly diaphragmatic, but its rate was approximately normal, except in one in which it was only 9 per minute, while the temperature remained under 80°F. It must be, however, remembered that when the respiratory movements are much restricted their rate is usually increased, and consequently we may assume a relative slowing of respiration.

The fourth special symptom was the small amount of urine passed. In one man who lived 48 hours no urine was secreted; another secreted only 20 ounces in 3 days; a third probably only 8 ounces in 4 days; while from a fourth, whose temperature varied between 87°F. and 105°F. and his pulse rate between 40 and 104, only 20 to 25 ounces could be drawn off during the first 3 days, but the daily amount

increased to 50 to 60 ounces for the last 2 days, when his temperature ranged between 100° F. and 105° F. The amount of urea was estimated in two cases in which very little urine was secreted, and its percentage was approximately normal.

The mental state of these patients was another interesting feature; when the temperature was very low, or at least below 85° F., they were stuporose or extremely lethargic, but, with the exception of one case, they could be roused, and then appeared quite intelligent and answered questions rationally, though they always tended to drift quickly again into a lethargic and apathetic state, unconcerned with their condition and immediate wants. As their temperature rose this mental lethargy quickly passed off, and they became bright and fully conscious of their serious condition—in fact, their mental state varied directly with their temperature.

The general appearance of these patients with low temperature, slow pulse rate, stupor or mental lethargy, and low metabolic exchange, as indicated by the small amount of urine of normal constitution secreted, reminded one strongly of an animal in hibernation, and as in the hibernating animal the pulse rate and amount of urine secreted increased and the stupor passed off as the temperature rose to normal limits.

Post-mortem examinations were obtained in nine of the ten cases; in two the chief injury was to the sixth cervical segment, in two to the seventh, in one the seventh and eighth cervical segments were damaged, and in the remaining four the eighth cervical and the first dorsal. In four of these cords there were practically complete transverse lesions; in the others the injury seemed to be only partial, but except in one of these a microscopical examination has not yet been made. Further, from the clinical signs a complete transverse lesion would not be diagnosed in at least three of these cases—in fact, one was able to move both legs from the time he came under observation till his death. In his cord microscopical examination revealed hemorrhages with œdema and foci of necrosis in the seventh and the upper part of the eighth cervical segments, small hemorrhages and swollen axis cylinders above the lesion as high as the fourth, and a cylindrical cavity descending through the dorsal columns from the first to the third dorsal segment.

A condition similar to that above described has been repeatedly produced in animals by section of the cervical cord; this is, in fact, followed by a fall in temperature and blood pressure, a slowing of the pulse and of respiration, diminished secretion of urine, and death within a few days. And the same symptoms have been observed in man when the cervical cord has been injured by a fracture or dislocation of the neck. Parkin, for instance, records a case of destruction of the fifth to the seventh cervical segments,

in which the temperature fell as low as 78° F. and the pulse rate to 26–37 per minute; and Nieden another case of dislocation of the first dorsal vertebra, in which the temperature gradually fell to 80.6° F. before death on the eleventh day, and the pulse rate sank to 30 per minute. The blood pressure was evidently very low in both these cases, as the pulse was described as hardly perceptible.

On the other hand, we have seen several cases of complete or very severe injury between the fifth cervical and the first dorsal segment in which these symptoms were not present.

We have some evidence that these patients do react to some extent as cold-blooded animals, and that their temperature may vary with the external temperature; in one patient, for instance, the temperature rose from 84.2° to 95° F. when he was placed on a hot-water bed and packed around with hot-water bottles, and another from 86° to 105.6° F., when he was brought into a room heated to 70° F. and also surrounded by warm bedclothes and hot-water bottles. This interpretation of the latter observation is not, however, beyond doubt, as a final rise of temperature occurred in other patients.

In another case an injection of pituitrin brought the temperature for a few hours almost up to normal, and increased the pulse and respiratory rates, while at the same time the patient, who had been very dull and lethargic, became temporarily bright and intelligent.

The interpretation of these observations can not be attempted here; they might be taken as evidence of the existence of a heat regulating center in the lower part of the cervical enlargement, as Dr. Hale White has assumed, or, on the other hand, the fall of temperature might be attributed to diminished heat production consequent on muscular inactivity; a similar fall occurs in curarized animals. Probably the most important factors are deficient thermogenesis and a visceral vasomotor palsy produced by shock in the sympathetic system. Capt. Shorten, however, in a short comment on the case published by Lieut. Oliver and Winfield, suggested that the symptoms may be due to the interruption of descending fibers in the cord which control and regulate the activity of the adrenals, and Prof. Harvey Cushing, who kindly saw one of our cases, had suggested previously to us that a cervical sympathetic palsy may disturb the functions of the pituitary and that this may interfere with the correlated activity of the suprarenals. If Capt. Shorten's hypothesis is correct we could expect to find hypothermia and its associated symptoms more commonly in cervical and bulbar lesions, and its absence after section of the cervical sympathetics argues against Dr. Harvey Cushing's suggestion. The adrenals appeared normal to the naked eye in one of our cases.

CERVICAL PYREXIA.

Benjamin Brodie, Chossat, and others have described a rise of temperature in animals after injury of the cervical cord, and Sir Jonathan Hutchinson, Sir Hermann Weber, and numerous other clinical observers have observed pyrexia with similar injuries in man. As in most of our patients there were septic wounds associated with the spinal lesion, and as in several cystitis coexisted, particular care is necessary in interpreting our observations on this point, but some are unequivocal. In one patient, for instance, with symptoms of a partial lesion in the fourth cervical segment and a small clean entry wound of a rifle bullet just below the tip of the right mastoid, in which there were no signs of infection, the temperature on several occasions rose to 104° F. and quickly fell again to normal. In this and in other patients the pyrexia was not associated with any signs of illness or discomfort, and the pulse rate did not vary as the temperature rose. In several other patients with partial or unilateral lesions between the third and sixth cervical segments, whom we were able to keep under observation for several weeks, the temperature was frequently above normal, and often reached 102° F. to 103° F. without any apparent cause.

Another interesting observation was persistent shivering of the shoulders, neck, and face without any rise of temperature of disturbance of the pulse rate, and without any subjective feeling of coldness associated with lesions of the lowest cervical and upper three dorsal segments. This occurred only in severe injuries of this region, and persisted over several days.

POLYURIA.

We have described diminished secretion of urine with lesions of the lower part of the cervical enlargement, but the daily quantity is frequently much increased when the injury lies in the middle of the dorsal cord, especially between the fifth and eighth dorsal segments. From one patient with a total transverse lesion between the seventh and eighth dorsal segments an average of 145 ounces was drawn off by catheter during the first six days he was under observation, even 215 ounces in one day. During the next six days and till death the average daily amount secreted was 90 ounces. In another patient with a severe lesion of the fifth dorsal segment the average amount drawn off during the first 18 days was 80 ounces; and in a third, who had sustained a complete destruction of the eighth dorsal segment, the daily average during the first three weeks was 125 ounces, but fell during the next three weeks to a daily average of 66 ounces. In fact, in the majority of patients with injuries to this region of the cord in whom observations were made, an excessive amount of urine was secreted. Unfortunately, we have not yet ob-

tained a complete analysis of the urine when it is in great excess.

This polyuria is probably due to paralysis of the sympathetic fibers, and especially of the vasoconstrictors, to the kidney. Claude Bernard and Sir John Rose Bradford, it will be remembered, produced it by section of the splanchnics and of the lower dorsal roots.

PULSE RATE.

While a slow pulse rate has been one of the characteristic symptoms of severe lesions of the lower part of the cervical enlargement, a marked and persistent acceleration of its rate was often present in partial lesions of this region, especially when the upper two dorsal segments were involved. In one case of partial destruction of the second dorsal segment, for instance, it rarely fell below 100 per minute and was frequently 120, and this independently of any rise of temperature or other obvious cause. In another severe injury of the same segment it ranged between 72 and 140 per minute, and in a third it never fell below 120 per minute. But the most striking alterations are perhaps seen with partial injuries of the four lower cervical segments in which the patient seems in perfect health apart from his spinal injury. In one such patient, who remained three months under observation with an originally slight injury of the fourth cervical segment which later progressed, the pulse rate constantly lay between 100 and 120, and only occasionally in the latter part of this period fell to 90 per minute, and in a similar uncomplicated case of injury to the fifth cervical segment the rate varied between 96 and 130 per minute. It was always regular, however, in these cases and of good volume and fair tension.

VOMITING.

When the mid-dorsal region is severely injured the abdomen is frequently tense and blown out and the patient presents the symptoms of paralytic distension of the intestines. But a more striking symptom which is sometimes associated with it, but which often occurs without any objective symptoms of abdominal disturbance, is vomiting. In the larger proportion of the cases in which it was observed the lesion lay in the sixth, seventh, or eighth dorsal segment.

It is often so persistent that it threatened life, as in severe cases no nourishment could be kept down. In several instances it set in within a few hours of infliction of the wound, and in many severe cases persisted till death or as long as we had the patients under observation. It was generally forcible and projectile, and was apparently associated with much discomfort, but with little nausea, though a few patients complained of the feeling of sickness. In mild cases it occurred only some time after taking food,

and this alone might be brought up in a half-digested state, but when severer it was more or less constant and mucous stuff occasionally colored with bile, and in a few instances bloodstained, was ejected. In some of our notes the similarity to the vomiting of a tabetic crisis is remarked on.

As in the large majority of cases in which this type of vomiting occurred the lesion lay in the region of the sympathetic outflow to the stomach it might be attributed to irritation or disturbance of the function of these fibers. And there is much evidence that this is its cause. Almost without exception these patients complained of girdle pains around the body, or on one or other side, between the level of the xiphoid and umbilicus, and of great tenderness to light contact, rubbing, and other stimuli in the region in which the referred pain and tenderness associated with gastric disease occurs. Further, in several there was a persistent local contraction of a portion of the abdominal muscles between the xiphoid and the umbilicus corresponding to the cutaneous hyperæsthesia, which increased and produced pain when this area of skin was stimulated. In fact, the firm resistance and tenderness of this area to touch occasionally gave rise to the suspicion of a large intra-abdominal lesion. In a few patients the intercostals innervated by the same dorsal roots were also in contraction, and everted the ribs to which these were attached.

Vomiting occasionally occurred after injury to other regions of the cord, too, but in most of these cases it was associated with and probably due to intestinal paralysis, to severe septic infection, or to the cystitis or pyelonephritis which occur so frequently with spinal lesions. In a few cases of cervical injury, however, it was a prominent symptom, and could not be attributed to any of these causes.

When severe and frequent, such vomiting naturally exhausted the patient and induced emaciation. Great loss of flesh also occurs, as might be expected, in severe cases which run a downward course, but it is an interesting fact that it was seen also in patients with relatively slight injuries of the cervical enlargement who took food well and even had excessive appetites. We have observed several patients, for instance, with unilateral lesions of this region producing the Brown-Séquard syndrome, but apparently not affecting their general health, in whom there was extreme emaciation. In some of these the pulse rate was increased, and there was slight pyrexia, but otherwise the visceral functions seemed unaffected.

PRIAPISM.

Numerous other symptoms occurred as a result of spinal lesions, to which time will not permit reference here. Priapism has been frequently described, especially with lesions in the cervical region, but we have observed it in only a small proportion of our cases, and it seemed to occur relatively as frequently with lesions of the dorsal as of the

higher segments. It was usually merely a soft turgescence of the penis. If, as is assumed, it is merely due to vascular engorgement, it is interesting to note that it occurred in two of our patients with low temperature, low blood pressure, and a slow pulse rate; it was present in one man when the blood pressure was only 73 mm. of mercury.

TROPHIC DISTURBANCES.

Various trophic disturbances were common in the severer cases, especially bullæ and blisters in those parts of the paralyzed regions which were subjected to any pressure. Irregular patches of red or strawberry-colored discoloration, which were scarcely modified by pressure and disappeared slowly leaving a slight mottling of the skin, also occurred frequently in the same parts. Joint changes were not common in the early stages, though there was occasionally effusion with the knees or ankles when the legs were completely paralyzed, and in a few patients we observed early arthritic affection of the fingers and wrist when the cervical cord was damaged.

HERPES.

Herpes occurred in nine cases, either immediately above or at the upper margin of the sensory loss. It developed between the third and fifteenth day, and in three instances recurred in the same area. It usually first appeared as a zone of diffuse erythema with small papules which later became vesicular or pustular, generally in a region in which there was either pain or tenderness. It usually lasted 7 to 14 days and disappeared, leaving some desquamation and brownish discoloration of the skin. Such larger vesicles as are seen in idiopathic herpes were not observed, and in one case there were only papules and vesicles without any erythema of the skin. In two cases in which post-mortem examinations were obtained the corresponding spinal ganglia were found bruised by displaced fragments of the vertebral column, and the clinical symptoms or the course of the missile made it probable that a ganglion was damaged in the other cases, too; its pathology is consequently allied to that described for idiopathic herpes by Bärensprung, and confirmed by Drs. Head and Campbell.

The state of sensation in the herpetic zone was variable; in some cases there was only excessive tenderness to contact, rubbing, cold, and other stimuli, associated with spontaneous pain; in other there were the symptoms of a root lesion; that is, a band of insensitiveness to pin prick and to moderate degrees of temperature, with loss or diminution of tactility within it. In those cases in which there was definite sensory loss it was found that this was not coterminous or did not correspond with the area of the eruption. Sherrington has shown that the dermatomeres are not superimposed in the myomeres, and it is obvious, too, that the pe-

ripheral distribution of the radicular fibers, which, when injured, are concerned in the production of herpes, do not coincide accurately with either.

III. THE SENSORY DISTURBANCES IN SPINAL INJURIES.

Nearly 60 years ago the first valuable contribution to this subject was made by Brown-Sequard, who from both clinical and experimental observations concluded that a one-sided lesion of the spinal cord produces motor paralysis, with hyperesthesia and loss of muscle sense on the same side, and in addition anesthesia of all other forms of sensibility on the opposite half of the body.

These conclusions have been, however, violently attacked by both physiologists and clinicians, and even Brown-Sequard himself later modified his views, but they have formed the basis of all subsequent contributions, and it has been, in fact, only within recent years that any important additions or modifications have been made to them.

The exact intraspinal course of the various forms of sensation soon began to excite interest. Many workers, following Brown-Sequard and Schiff, held that pain and temperature at least pass upward through the gray matter, while tactile impressions and those that underlie the sense of position ascend, according to Schiff, through the dorsal columns. Bechterew's and Bikel's experiments, however, make it probable that impressions of pain are conducted after decussation through the white matter of the ventral part of the lateral columns. But it is obvious that neither the grouping nor the course of sensory impulses within the cord could be determined by experiments on animals, and it is not surprising that the most reliable conclusions we have were obtained from clinical observations on suitable cases of injury or disease in man. It is largely to Petren that we owe the most accurate and comprehensive summary of clinical observations. From the collation of a large number of cases he has concluded that impressions of pain and temperature pass through the opposite lateral column, "muscle sense" through the homolateral dorsal column, while two paths are open to tactile impressions—one through the uncrossed exogenous fibers of the dorsal column, another in the opposite lateral column. A careful and elaborate analysis by Drs. Head and Thompson of sensory disturbances produced by spinal lesions confirmed these conclusions and showed that when the appreciation of cutaneous pain is lost that produced by pressure is also disturbed, that light touch and heavy touch are lost simultaneously, and that the appreciation of all degrees of temperature is abolished together. They also found that the ability to recognize the simultaneous contacts of two compass points depends on impulses conducted through the homolateral dorsal column. The affection of other forms of sensation by spinal lesions has been also investigated. The French school particularly has been

interested in the appreciation of the vibrations of a heavy tuning fork, and Egger, as well as Seiffer and Rydel, have shown that disturbance of this is generally associated with loss of the sense of position, and that it consequently depends on the integrity of the dorsal columns. Finally, working with Dr. Head, I found that in unilateral spinal lesions the appreciation of weight and the recognition of size and shape are frequently lost in the paralyzed side, and from this observation, correlated with other facts, we concluded that the impulses that subserve these functions also ascend uncrossed through the dorsal column; we also confirmed earlier observations that the appreciation of vibration passes by this path, too.

The nature of our material, and especially the fact that we have been able to investigate most of our cases only in that early stage in which certain symptoms may be attributed to shock and other to partial lesions and incomplete blocking of the passage of sensory impulses, makes extreme caution necessary in drawing final conclusions on the mode of sensory conduction within the cord, but the disturbances we observed must have an important bearing on this subject. We have been, however, able to keep certain cases under observation as long as 10 to 12 weeks after the infliction of the wound.

REMOTE SENSORY DISTURBANCES.

Anaesthesia to pain and temperature is certainly the most common and prominent disturbance of cutaneous sensibility; in incomplete transverse lesions, for instance, it is frequently found that though tactile stimuli can be appreciated, pricking or the application of heat or cold, even of extreme degrees and over a large area, evoke no sensation apart from that of contact. Both are always lost on the side opposite to the lesion when this is unilateral and lies above the first lumbar segment, and more diminished on this side when the injury is bilateral, but more severe on the one side. This crossed relation does not occur when the lesion lies below the twelfth dorsal segment. During recovery from bilateral lesions, too, tactile sensibility usually returns before that to painful or thermal stimuli. As a rule both these forms of sensation were lost together, but in some cases only thermal appreciation was disturbed.

We have not yet observed a case with primary isolated affection of either heat or cold, but during recovery sensibility to either may reappear some time before stimulation with the other evokes any sensation. Some dissociation is, however, common at the upper level of the remote sensory loss, but this will be discussed later when the mode of decussation of the sensory paths will be considered. Many cases seen confirm the conclusion put forward by Drs. Head and Thompson, that when pain can not be excited by cutaneous stimuli it can not be produced by excessive pressure; we have, however, seen cases in which heavy pressure

gave pain the parts which were completely anagesic to pin pricks, but it must be admitted that in some of these at least sensibility to prick returned within a relatively short period. Their further conclusion that sensibility to all degrees of temperature is affected simultaneously may be accepted if it is borne in mind that when the disease is slight the appreciation of moderate stimuli only may be lost; everything is, however, in favor of their view that thermal stimuli of all degrees pass by the same intraspinal paths, but that, as the study of syringomyelia shows, heat and cold are conducted by separate fibers.

Remote tactile anesthesia was present in only a very few of our cases of unilateral spinal lesion, and then it generally corresponded with the analgesia—that is, it occurred on the side opposite to the injury. Frequently, however, especially when cotton wool was used as a stimulus over hair-clad parts, there was a marked subjective difference in the sensations evoked on the two sides, as contacts on the analgesic area “tickled less” or were “smoother” than on the side of the lesion; this might be, on the one hand, attributed to the homolateral hyperesthesia or unnatural sensitiveness to various stimuli which Brown-Sequard originally described, but when the sensations were compared with those similarly evoked from normal parts it was evident that the tickling element in the sensation was defective on the contralateral side. On the other hand, this difference was not due to a diminution of tactile acuity, as this could not be demonstrated when other stimuli were employed; in a few cases in which Von Frey’s hairs were used no definite threshold difference between the two sides was found. Further, on stroking the sole, or on applying any other stimulus which naturally tickles, less reaction was evoked on the side opposite to the injury. It appears, therefore, that of the two spinal paths open to tactile impulses only the crossed fibers which are closely associated with those that carry pain are concerned in the conduction of the affective impressions produced by many tactile stimuli. This observation is particularly interesting, as lesions of certain portions of the optic thalami show that at this level the impulses that underlie tickling are closely related to those of pain.

But though crossed tactile anesthesia was the rule in our cases, in a few there was greater diminution of sensibility to light touch on the side of the lesion, or on the side of the greater motor paralysis; even homolateral anesthesia with crossed analgesia occurred in one case.

Another interesting form of dissociation of sensation was represented by a case of incomplete injury due to fracture of the left side of the sixth cervical vertebra by a rifle bullet. There was complete paralysis of the left leg and of the trunk muscles on this side, and all the movements of the opposite limb were weak; on the right side there was complete loss of sensibility to touch, pain, and temperature, while on the less paralyzed half of the body pricks and other painful stimuli

were appreciated normally, and thermal sensation was only slightly diminished, but there was complete anesthesia to both light and heavy touches. The sense of position and the appreciation of vibration were completely lost on both sides below the level of the second rib.

In such a case we have the converse of the general rule that tactile sensibility suffers less severely than sensation to pain.

One interesting question that arises from these observations is the mode of decussation of the sensory fibers of the second order which convey impressions of touch, pain, and temperature, and the obliquity with which they cross to the opposite side of the cord. In cases in which the lesion was unilateral the upper limit of the remote anesthesia to these different forms of sensation varied, and as a rule the upper border of the contralateral loss did not correspond to the segmental level of the injury. If such observations were sufficiently numerous it would be obviously possible to determine the number of segments necessary for the complete decussation of each set of fibers of specific sensory function. To obtain unequivocal results, however, it would be necessary to consider only cases in which the lesion interrupted these fibers after their decussation is completed and in which their interruption is total; unfortunately such cases are rare.

In the mid-dorsal region the crossing of pain and thermal impulses apparently occurs quickly, and is probably complete about one segment above the entry of the dorsal roots that carry them to the cord. Thermal impressions probably cross here less rapidly than those of pain, and as touch, if it is affected, is generally lost to the slightly lower level, the fibers of the second order that convey it probably require two segments for decussation. This conclusion is accepted with full recognition of the fact that the peripheral overlap of the tactile root fibers is greater than that of those concerned with pain and temperature. The same order holds for the upper dorsal segments, but here the obliquity of decussation is greater; pain and temperature impressions do not cross for at least two segments after their entry into the cord, and three are frequently required.

The higher we go in the cervical enlargement the slower does the decussation become. At the fourth cervical segment, for instance, the decussation of the pain impulses is not complete till five to six segments after their entry through the dorsal roots, and that of thermal impressions for four to five segments. In the cervical enlargement pain seems to cross within about four segments, thermal sensibility within three to four, and touch somewhat more obliquely than pain; as the upper margin of the anesthesia to heat is generally slightly higher than that to cold it may be assumed that the afferent impulses that subserve the latter decussate more slowly.

When the lesion is not complete, and especially if it is unilateral, the upper border of the sensory loss frequently

retreats caudalwards. In one case, for instance, the upper margin of the complete analgesia altered from the sixth cervical to the ninth dorsal root area in two months, and in another case of partial bilateral lesion it retreated from the upper margin of the right cervical root area to the level of the umbilicus within four weeks. A parallel recovery of thermal sensibility may be frequently observed, but it is usually much slower, and heat and cold are then frequently dissociated, the latter recovering, as a rule, earlier than sensation to heat; but the converse may occur. On the other hand, even when the lesion lies high in the cord, and has produced total or partial loss of sensation to the corresponding level, it is not uncommon to find the area of the lower spinal roots, and especially the skin in the region of the anus, sensitive to one or other mode of stimulation. Further, during recovery it is sometimes in these regions in which sensation first reappears. In a case of injury of the fourth cervical segment, for instance, which produced thermal, pain, and tactile anesthesia to the root of the neck, prick could be well appreciated on all of the sacral and on the fifth lumbar root areas on the left side. And in another the third, fourth, and fifth sacral areas escaped, though there was otherwise total loss of sensation to the level of the umbilicus. There is generally a remarkable dissociation of sensation in this caudal area; occasionally only pain can be appreciated, but it is frequently sensitive to touch, too, and either to heat or cold, or to both. The area sensitive to thermal stimuli is, however, generally smaller than that in which pain can be felt. As a rule the caudal anesthetic part in such cases corresponds roughly to areas of root distribution to the skin.

These two phenomena, the caudalward recovery of sensation and the escape or early reappearance of sensation in the caudal areas, throw light on the arrangement of the sensory fibers of the second order as they ascend through the ventrolateral columns. They indicate a lamellar arrangement in which the fibers that carry any specific form of sensation from successive dorsal roots lie in series; and as there is a general law that the longer ascending fibers lie nearer the periphery of the cord, those that convey impressions from the lower spinal roots are probably placed lateral to those that have later reached the contralateral side. The escape of the sacral root areas would therefore indicate a lesion that involves only the more mesial fibers of the sensory path, while an anesthesia disproportionately low in relation to the level of the spinal injury would suggest a local destruction of its more lateral fibers. When it becomes possible to correlate the exact histological changes in these cases with the results of careful clinical examinations, definite conclusions on the exact course of the fibers that carry various forms of sensation from different regions of the body will be possible.

Microscopical examination has shown that extensive softening and even secondary changes occur frequently in the

center of the cord, especially in the cervical region, and we might consequently expect to find sensory disturbances resulting from the interruption of the fibers that decussate at the level of such a lesion; certain of the sensory changes that occur in syringomyelia are due to this cause. The most striking example of this condition that we have seen was:

SUBJECTIVE SENSORY SYMPTOMS.

Subjective sensory symptoms were not uncommon; pain, for instance, is very commonly present in the parts that correspond to the segmental level of the spinal injury, but here it may be due to irritation of the dorsal roots, or may be only associated with the hyperesthesia which frequently occurs in this region. Distant pain—in the leg, for instance, when the spinal wound was in the cervical region—occurred, however, in a certain number of cases and can be attributed only to the spinal lesion. It was frequently only transient or disappeared within one or two weeks, but occasionally persisted as long as we had the patients under observation, in one case for two months. It was generally described as a burning or an aching pain, sometimes only as a numbness or tingling, which increased when the part was touched or handled. In one patient it was so severe that it prevented sleep, and needed morphine. It was generally most severe at night, and in some instances increased when the part was exposed or moved, though there might be no demonstrable hyperesthesia. It sometimes spread over the whole side of the body below the level of the injury, but it was frequently limited to the foot or leg. It occurred only with relatively slight or unilateral lesions, and in the latter cases was always on the same side as the injury and on that on which there was no cutaneous sensory loss. In a few instances, however, no sensory loss could be discovered in either side, or if any existed, it was only a slight diminution of sensibility to pain and temperature.

The condition known as hyperesthesia was extremely common; it may occur, as was originally described by Brown-Séquard, on the side of the injury when there is a unilateral lesion; or on areas which had been anesthetic as sensation recovers, or even more commonly as a band on one or both sides of the trunk or down the limbs, at the upper margin of the sensory loss.

A homolateral hyperesthesia in Brown-Séquard cases is by no means constant, though we observed it occasionally. It extended over the whole half of the body almost up to the segmental level of the injury. Here pin prick, heavy pressure, and especially scraping or even rubbing hair-clad parts with a wisp of cotton wool produced severe pain and much more reaction than these stimuli on normal parts. The application of cold, too, usually evolved pain, and heat of 45° C. and upward caused a severe burning sensation. In one case pin prick not only gave more discomfort than normal, but this persisted abnormally long. This distant hyper-

esthesia always showed, however, a tendency to diminish rapidly and disappeared in some cases while they were under our observation. Its pathogenesis has been already the subject of much discussion and experiment. The hypothesis that it is due to section of efferent inhibitory fibers seems very improbable, while the fact that it is generally transient or disappears rapidly is more in favor of the view that it is due to an inflammatory reaction, or rather to oedema and other such changes, at the site of the lesion than to overloading to the afferent tracks that remain open to sensory impulses.

An excellent example of hyperesthesia in areas in which sensation is recovering was a case in which, owing to a transverse wound across the back of the neck which fractured the sixth and seventh cervical spines as well as the lamina of the sixth vertebra, a complete paraplegia with analgesia and thermoanesthesia to the level of the eighth cervical root areas set in at once. Within a fortnight, however, there was complete pain loss only below the umbilicus, and the appreciation of thermal stimuli was only diminished above this level. In the area in which sensation had recovered pricks produced an extremely unpleasant burning sensation, and high and low degrees of temperature gave much more discomfort than on normal parts. He later came under the care of Dr. Head, who found the same condition more than two months after the infliction of the wound.

Finally we come to the hyperesthesia which is so commonly found at the upper level of the sensory loss on one or both sides, with either complete or partial lesions. On the trunk it is generally associated with a girdle sensation of pain, burning, tingling, or constriction, and on the limbs with pain or paresthesia over the areas of one or more dorsal roots. It may persist for weeks, but usually diminishes. When severe the lightest contact or even the approach of anyone to his bedside may be feared by the patient, and the movement of his bedclothes or the removal of his shirt or bandages may excite severe pain. Local muscular contractions are frequently associated with it. The state of sensation in the hyperesthetic area has been carefully worked out in several cases; it is found to be variable. No loss to light contact was ever found, and when they were used no raising of the threshold was discovered to Von Frey's hairs, but in three cases contact with No. 4 and No. 5, which only gave a maximal threshold reading on normal parts, produced a sharp stinging or burning "like a red-hot iron" on the hyperesthetic zone. Pain was most readily produced by any moving stimulus, as a wisp of cotton wool or by scraping with any rough or sharp object, but, on the other hand, moderate pressure generally gave no discomfort.

The state of sensibility to pain and temperature varied; in one group the threshold to pain was, if anything, diminished, while that to temperature was unaffected, but any degree of cold and heat above 45° C. evoked much pain. In the other class sensibility to pain and temperature was lost

or diminished over part of the area that was painful to rubbing or scraping.

The origin of this hyperesthesia is interesting; it is most commonly attributed to irritation of the corresponding dorsal roots, and this seems to be frequently the actual cause, but there are many facts which prevent us from accepting this explanation in every case. It is often, for instance, much more extensive than it would be if due to irritation of even two pairs of roots in the neighborhood of the wound; in one case of injury to the spine of the axis it extended over both upper limbs and to the level of the nipples, and in another patient in whom the fourth cervical vertebra was damaged it spread not only over both arms but to the base of the xiphoid. But an even stronger argument is the fact that in cases of the Brown-Sequard syndrome, in which there were both hyperesthesia of the homolateral side and a local area of pain and tenderness at the level of the lesion, no essential difference may be found between them. It is therefore probable that in many cases at least the cause of the pain is to be found within the cord, and that it is due to edema, circulatory disturbances, or slight diffuse lesions, as Brown-Sequard originally postulated as the explanation of the local contralateral hyperesthesia he described. Finally, in several cases we have found no injury, on post-mortem examination, to the roots which corresponded to the areas that were hyperesthetic.

PROGNOSIS.

The prognosis during the first two weeks in any one case is extremely difficult, and it must be admitted that there is no one sign or symptom from which we can draw reliable conclusions on the severity of the lesion, or from which we can say, when there is complete motor and sensory paralysis, as there nearly always is in the earliest stages, whether the cord is completely divided or not. It must be remembered that though neither the cells nor the fibers of the spinal cord do regenerate, very considerable improvement may occur, as at least part of the early symptoms are due to edema, circulatory disturbances, and to incomplete damage. The structural damage is consequently not always parallel to the functional loss. We have seen that the knee jerks are absent for a time with lesions of all degrees of severity, and this consequently can not be a guide in prognosis. The most reliable information is perhaps given by the state of tone in the muscles of the lower limbs; after three or four days the legs are generally very flaccid and their muscles toneless when the lesion is severe and irrecoverable, and gradually become more so and waste. The preservation of tone in the muscles is, on the other hand, an indication that some improvement may be expected. Valuable information can be also obtained by stimulation of the soles, as the amount of reflex movement that results varies more or less inversely with the severity of the injury. When this is complete, no

reflex muscle contraction can be, as a rule, elicited, while in all stages of slighter damage a brisk withdrawal reflex can be obtained.

Probably no serviceable recovery can be expected if the plantar responses are flexor.

In less severe cases, in which all forms of sensation are not abolished, the amount of disturbance of the latter is an indication of the amount of the cord damaged; we have generally seen the promise of useful recovery when tactile stimuli could be felt in the lower limbs within the first two or three days.

When the cervical region is injured the upper limbs are usually more paralyzed than the lower, and remain flaccid and waste while these show signs of recovery; histological examination shows that this atrophic palsy of the arms is due to extensive softening of and hemorrhages into the ventral horns, and as the motor cells contained in them are readily destroyed the chance of much improvement is slight.

If recovery sets in early, steadily progressive improvement may be, however, expected, unless complications occur. We know of a few patients, however, in whom the symptoms increased after movement, as Case IV, who lost again the power of movement he had regained in his right leg during his transference to England; and in one other case we observed syringomyelia develop some time after the infliction of the injury.

TREATMENT.

Owing to the nature of the lesions the treatment of these spinal injuries is naturally unpromising. The damage to the spinal cord is done when the wound is inflicted, and we are unable to influence it by treatment. In many cases surgical intervention and the removal of missiles or displaced bone which compress the cord have given a hope of greater recovery, and should be attempted if the symptoms or an X-ray examination make it probable that the cord is compressed, and that there is any prospect of recovery. But it must be realized that in such cases the symptoms are certainly more dependent on intramedullary changes produced at the time rather than on compression.

Dr. A. R. Allen showed experimentally some years ago that the symptoms produced by severe contusion of the cord can be relieved and recovery made possible by incising the dorsal columns at the level of the injury, thus draining away oedematous fluid and intramedullary hemorrhages, and allowing the swollen fibers to expand, but it is necessary that this operation should be performed within a few hours of the infliction of the injury. This is rarely possible in warfare, and the early symptoms are so equivocal that if resorted to more harm than good might easily be done.

A large proportion of cases of spinal injury die soon after the infliction of the wound from shock or associated wounds of the chest or abdomen. Among those that survive the greatest danger is from cystitis and pyelonephritis and the

development of extensive bedsores. A large part of the responsibility consequently falls on the nursing. When cystitis is threatened or has developed we have seen excellent results from suprapubic drainage. Finally, the danger of moving the patient must be borne in mind; the risk is obvious if the vertebral column is fractured and if detached pieces of bone lie within the canal transit. Further, we have evidence that secondary changes are more liable to develop after movement; absolute rest is consequently advisable during the first few weeks if the symptoms hold out any prospect of useful recovery.

James Collier: Gunshot Wounds and Injuries of the Spinal Cord.
Lancet, Apr. 1, 1916, p. 711.

THE NATURE OF THE LESIONS.

The lesions that Collier met with were caused by high-velocity bullets, shrapnel, fragments of shell casing, and by the concussion of high explosives without any external wound. A classification of the lesions, though arbitrary, is useful in considering the mode in which they are produced. Often more than one kind of lesion is present. The following is a rough classification: (1) Direct lesions. Any lesion resulting from the passage of a missile across the spinal canal, whether it touch the spinal cord or not, is a direct lesion. (2) Indirect lesions: (a) Those due to the indriving of bone, etc., into the spinal canal. (b) Impact lesions where the missile strikes against the bony wall of the spinal canal. (c) Concussion lesions from the shock of high explosions. (3) Secondary lesions: Perithecal and intrathecal hemorrhage, medullary hemorrhage and thrombosis, meningitis, edema. These lesions are important as the cause of the deepening of symptoms, often at a considerable time after the injury. (4) Remote lesions which may be found anywhere in the spinal cord and chiefly near the surface: Spots of necrosis, sievelike rarefaction, punctiform hemorrhages, edema, swelling of axons. These lesions are produced by the sudden raising of the intraspinal pressure caused by the passage of the missiles through the intraspinal space or by a general concussion effect. They are well marked in cases of concussion without external wound.

The mode of production of the *direct injury* is clear. The passage of a fast-moving projectile through a tissue causes not only a heat effect generated by the friction but an intense raising of the pressure in the tissues for some distance round the track, and if the bullet cross the spinal canal, which contains a fluid pressure, this momentary and severe pressure effect is transmitted to the whole of the nervous system confined within the cerebrospinal space. The cerebrospinal space being an elastic cavity the increase of pressure caused by the passage of the bullet will be greatest at the site of the track and will decrease in proportion to the distance from the track, but will be greater below the site of passage than above it, because the lower portion of the spinal canal is more closed and therefore less elastic. The damage

to the spinal cord even when the bullet does not touch the cord nor lacerate the membranes is therefore greatest where the bullet has crossed, but it extends for a considerable distance above and below this. Lesions of the cord in places remote from the chief seat of injury are not infrequently found which are attributable to this increase of pressure, and these are always more marked in the distal part of the spinal cord. The intracranial effect of this increase of pressure is the immediate loss of consciousness which is sometimes met with, and more often in cervical injuries than in those lower down. Possibly the initial loss of consciousness may be an index that the bullet traversed the spinal canal. This certainly holds good for the cervical region, for in three of my cases of cervical injury where consciousness was not lost, although the initial symptoms were severe, complete recovery occurred. It seems impossible that a bullet can cross the spinal canal without causing the most severe and irreparable of transverse lesions, and one may argue that in all the cases where relative or complete recovery occurs the lesion has been an indirect one—either an impact lesion or due to the indriving of bone. It is likely that a bullet does not so often cross the spinal canal, but is more often deflected by the bony wall.

Mode of production of impact lesion.—The passage of a high-velocity bullet in the immediate vicinity of the spinal canal or the impact of a projectile upon the bones forming the spinal canal may cause lesions of the spinal cord sometimes very severe notwithstanding that the walls of this canal remain intact. How are these local concussion lesions produced? The anatomical relations of the spinal cord practically forbid any such displacement of the cord within the canal as could bring it in contact with the bony wall and so bruise either directly or by contrecoup. Col. Gordon Holmes has shown that these impact lesions show their greatest intensity immediately under the point of impact and closest to the bullet track, as the case may be. The following is an explanation of the way in which these indirect local lesions of the cord are produced. The contents of the spinal canal which intervene between its parietes and the spinal cord, and made up of the perithecal fat and vessels, the theca and the cerebrospinal fluid, may be regarded as fluid, and the pressure within the spinal canal as a fluid pressure, contained in a vessel which has one rigid wall, the spinal canal, and one elastic wall, the compressible vessels of the cord, etc. Any lines of force either from excessive vibration, from impact, or from compression transmitted from one spot in the rigid wall will have their most intense effect upon the nearest point of the elastic wall, i. e., the nearest spot in the spinal cord and little elsewhere, the force becoming rapidly spent upon the elastic wall as it radiates. The damage to the spinal cord produced in this way by indirect injury may be from the most severe, with symptoms of a total transverse lesion with no recovery, to the slightest, with complete recovery.

Concussion lesions.—Severe indirect injuries of the spinal cord may occur from the bursting of high-explosive shells when the back is turned toward the force of explosion without any external wound occurring, without any detectable lesion of the bones, and even without bruising of the soft tissues.

In such cases as these, where severe spinal lesions have been produced by the force of high explosion at a distance without any external wound, it is possible to conceive that there has been such distortion of the spinal column as might produce a subluxation of the vertebræ which became immediately reduced, and that during this subluxation the bony rings which make up the spinal canal crossed to such an extent as to produce a severe local transverse lesion of the cord. That such a subluxation is possible has been proved experimentally upon the cadaver, and that it can severely pinch the spinal cord has also been proved, and there is much evidence in the records that such a subluxation, immediately reduced, is one of the lesions that may occur in cases of broken back. Collier examined one such case in which a total transverse lesion resulted from a fall from a haystack. The spinal cord was almost severed within the membranes, and although he macerated the bones he could find no sign of fracture. He did not, however, see that there was any evidence in favor of the occurrence of such a subluxation in the cases under consideration, and the histories of the above cases and of several others. We must therefore regard these lesions as impact lesions. It seems remarkable that the impact of the force of the explosion upon the dorsal surface of the body could be sufficient to produce local spinal concussion and a sharply local lesion of the cord without any evidence of severe bruising of the skin and soft tissues. In his three cases and in several others that he had heard of, the lesion was in the lower dorsal region. It is a point of great interest **as to whether such severe local lesions from explosion occur in other regions.**

Root lesions.—Sometimes the lesions produced by projectiles affect the spinal roots after they have left the thecal space to a much greater extent than they affect the spinal cord and thecal contents. This seems to be the case when there has been much fracture and crushing of the bone, and especially in the region of the transverse processes. There is usually much swelling and edema in the injured region of the spine. Such root lesions are not entirely due to direct injury, for they may be widely spread when the injury is comparatively local. They may result from subperiosteal hemorrhages and periosteal swelling, which strangle the roots in the intervertebral foramina; or from pachymeningeal hemorrhage.

Intrathecal hemorrhage.—Among the many lesions of the spinal cord met with resulting from bullet wounds there are two which merit special comment. The first of these lesions is intrathecal hemorrhage. Blood effused into a free thecal space finds its way with the stream of cerebrospinal

fluid into the lower part of the thecal space around the cauda equina and lumbo-sacral enlargement, and if massive in quantity may distend that space, causing pressure upon the roots and epiconus, and clotting there may cause such matting and cicatrization as may completely destroy by pressure and evascularisation the caudal equina, and if it extend high enough the lumbar enlargement as well. Smaller effusions by clotting and the formation of condensing adhesions may cause local signs of nerve-root involvement. In a severe condition of this kind where there has been extensive intrathecal hemorrhage—due, let us say, to a lesion of the mid-dorsal region—the physical signs in the lower extremities may resemble exactly those due to a complete lesion of the cauda equina or of the lumbo-sacral enlargement. In the less severe condition the local deposit and subsequent cicatrization of blood clot round individual roots of the cauda may cause physical signs difficult of explanation unless the possibility of the occurrence of intrathecal hemorrhage and its results are kept in mind.

It is easy to conceive of cases of thecal hemorrhage where the matter round the cauda equina is less severe and gives rise to partial lesions of the cauda only, and therefore in cases where the bullet lesion of the spinal cord is above the lumbar enlargement and unusual conditions of reflexes, spasm, and contracture are present in the legs, it is well to consider whether a condition of thecal hemorrhage and matting of the cauda equina may not exist. Local wasting of muscles and local loss of faradic excitability may be indications that such a condition exists. The early diagnosis of this condition in such a stage as to allow of the removal of the blood by lumbar puncture and washing out with citrate solution is apparently impossible, for the initial aspect of nearly all the spinal lesions resulting from bullets is that of total transverse lesion, and therefore the development and deepening of the signs of involvement of the cauda equina can not be detected.

REFLEX ACTION.

It is usually held that the initial condition of reflex action after the occurrence of severe lesion of the spinal cord is that there is complete loss of both jerks and superficial reflexes, or that if the loss is not complete the only sign of reflex action left is a feeble flexor response occurring in the toes, only obtainable from plantar stimulation, and that the early presence of the extensor response or its early return is indicative that the lesion is not complete and is of good prognostic import. Col. Gordon Holmes has laid it down very clearly that the condition of the plantar reflexes in all the cases which he has examined shortly after the injury has been either that they are completely absent or that there is a reduced flexion response, but he is careful to add that his examinations have taken place not earlier than the second day after the injury.

There may be four consecutive stages in the condition of the plantar reflexes following a transverse lesion of the cord: (1) An initial extensor response; (2) either a complete absence of any reflex, which may be the result of shock or of isolation alteration, or a reduced flexion reflex which is the result of isolation alteration which may come on rapidly; (3) the extensor response, which, when persistent, is indicative of a less severe lesion or alternatively of more recovery than the reduced flexion reflex; (4) the normal flexion reflex which returns when recovery is complete. The condition of the plantar reflex is therefore an index of the severity of the damage to the spinal cord and an important early indication as to whether recovery is occurring or not.

Col. Gordon Holmes has made the very interesting suggestion that the simple flexion of the toes, which can be obtained only by stimulating the sole in cases of severe transverse lesions of the cord, and which Collier has called the "reduced flexor response," is a unisegmental reflex involving the first sacral segment only, and that when the physiological vitality of the lumbar enlargement is much reduced by isolation from the higher nervous system the only reflex response that can be obtained is a unisegmental reflex. Collier's observations incline him to support strongly Col. Gordon Holmes's theory. He has repeatedly noticed, in a case where some recovery is occurring and where the reduced flexor response is changing to the extensor response, that a minimal stimulus will produce flexion of the toes and a more severe stimulus will result in extension of the toes, or that there is first flexion and subsequent extension of the toes, but never the reverse. That is to say, the threshold for the unisegmental reflex is lower, as one would expect.

Another most important point that the experience of this war has brought out is that the influence of the higher nervous system upon the reflex action and muscle tone of the spinal cord is strictly homolateral so far as the cord is concerned. That is to say, that a total unilateral lesion of the cord produces complete flaccidity with loss of knee and ankle jerk and reduction of superficial reflex upon the side of the lesion. This will explain those cases in which we meet with an extensor response upon one side and a reduced flexor response upon the other, in that one has a transverse lesion more complete upon one side than upon the other.

Contracture is a phenomenon which is intimately associated with the condition of reflex action. In these paralytic cases with which we are dealing there are three conditions of contracture of the feet: (1) The dropped foot with retracted toes. This is the ordinary pes cavus of spastic states—the crystallization of the extensor response. (2) The retracted foot with retracted toes. The calcaneus position is often extreme. The ankle jerk is always lost and the anterior tibial jerk marked. An extensor plantar reflex is always present. (3) The dropped foot with dropped toes, the position being similar to that of peripheral

neuritis. The plantar reflex is either absent or there is a reduced flexor response.

Collier has observed in several cases the dropped foot with dropped toes gradually changing into the spastic pes cavus with a change of the plantar reflex from the reduced flexor to the extensor type in cases where some improvement has taken place, and therefore considered that the dropped foot with dropped toes indicates a more complete transverse lesion and that it is produced by a relatively greater muscular tone in the flexors of the toes than elsewhere, and that it is associated with the reduced flexor response just as the pes cavus is associated with the extensor response in less severe transverse lesions.

The calcaneus position with retracted toes is a very remarkable phenomenon. It has been persistent in those cases in which I have met with it, and has been always associated with loss of knee and ankle jerk and increase of anterior tibial and hamstring jerks. The extensor response and active withdrawal reflex have always been present.

Collier carefully examined the electrical excitability of the calf muscles and of the anterior tibial muscles in extreme cases of the equinus and calcaneus positions, and found that there is no difference in the faradic excitability of these muscle groups, thus proving that these positions of feet and toes are not due to any secondary lesions of the cauda equina or lumbo-sacral enlargement.

He met with one curious reflex phenomenon in the leg in a way comparable to the curious reflex which Col. Gordon Holmes has observed in the arm in a case of cervical lesion. His case was one of a mid-dorsal lesion, total upon the right side of the cord and severe upon the left. The right leg was completely flaccid and the only sign of reflex action present was that on stimulating the sole, after a latent period of four seconds or more, a feeble flexion and adduction of the thigh occurred. This reflex was easily tired out, and was only obtainable on gentle stimulation of the sole.

The physical signs obtaining in the lower limbs in transverse lesions of the cord seem to be the same whatever the level of the lesion above the lumbar enlargement.

There is one question which he put to each of the speakers who followed him: What is the ultimate condition of the paralyzed region in cases of total transverse lesion of the cord which are long survived? His own suggestion was that the slow process of physiological deterioration which we call "isolation dystrophy" may reach such a point that no signs of activity of the lumbar enlargement remain.

To the usual types of paraplegia resulting from lesions of the spinal cord and representing different degrees of isolation of the distal segment, namely, "paralysis in extension" and "paralysis in flexion"—we might speak of a third type—that of complete flaccid paralysis. For it is from this stage of complete flaccid paralysis that the other types successively develop when improvement is occurring,

and to this type they successively return when there is an increasing lesion or from long-standing isolation of the distal segment. There is, of course, no sharp separation between these types; they merge into one another gradually.

DISTINCTION BETWEEN ROOT LESIONS AND CENTRAL LESIONS.

The diagnosis between lesions of the roots and lesions of the central gray matter is sometimes impossible, as, for example, when a lesion of either of these structures is complete. It is sometimes quite simple when a single root or so is damaged and there is characteristic sensory and motor loss in the area of distribution of the damaged root. In many cases, however, the diagnosis is far from a simple matter, and in some of these both lesions may exist together. The following points may be useful in the diagnosis: (1) In the cervical region extensive root lesions are only met with when there is severe injury of the bones, especially of the transverse processes and spines, and these are recognizable by deformity, swelling, and edema, and radiography in the absence of these signs, an atrophic palsy of the arms is probably the result of a central lesion, if it is extensive. (2) In central lesions the upper limit of the sensory loss is a line more or less transverse to the axis of the limb—that is to say, the sensory loss is of the “glove” or “stocking” pattern, in contrast to the more or less longitudinal limitation which obtains in root lesions. (3) In the cervical region a relative escape of the long columns or early signs of recovery in these with severe paralysis of the upper limbs is in favor of a root lesion, and may suggest the correct diagnosis at an early stage. (4) In the lumbo-sacral region the most certain indication is the level of the wound of the spine if this can be determined with certainty.

DISTURBANCES OF SENSIBILITY.

The chief points of interest in this connection are the paths taken by the sensory fibers in their spinal course, the sensory loss resulting from unilateral lesions, and the explanation of remote pains. The usually accepted paths of sensation in the spinal cord are as follows: (1) The path for touch is a double one, in the homolateral dorsal column and in the crossed lateral column; (2) pain, heat, and cold are conveyed by separate fibers in the crossed ventrolateral column; (3) sense of position, of passive movement, of vibration and recognition of form, and of the compass points are conveyed in the homolateral dorsal column. The crossing of the sensory fibers in the cord occupies one to two segments in the lower dorsal region, but increasingly more segments as the cord is ascended, so that in the cervical region some five segments are occupied by the crossing. There is no crossing below the last dorsal segment. The path for painful and thermal stimuli crosses more rapidly than

does touch. Consequently a unilateral lesion of the cord does not produce a Brown-Séquard syndrome when the lesion is at the level of the first lumbar segment or below this.

The symptoms of the unilateral lesion are that there is motor paralysis, loss of sense of position and of passive movement, and of appreciation of vibration, form, and compasses upon the same side below the lesion, and loss of pain, heat, and cold upon the opposite side. The bilateral loss in the region of the lesion from the involvement of both right and left crossing fibers is narrower for pain and temperature than for touch. This bilateral loss is much narrower in the dorsal than in the cervical regions, on account of the increasing obliquity of the crossing fibers. Collier's observations have been in accord with the above scheme with one exception. In the majority of his cases of unilateral lesion of the cord the vibration sense has been lost or most affected upon the opposite side, and this has obtained both in severe and in slight lesions. He must admit that he has no pathological evidence in such a case.

Remote pains are very obstinate in some cases. Their mode of origin is obscure. Obviously, they are only met with in those cases where the sensory conducting tracts are relatively intact. The usual symptom-complex that he has met with is that there is a transverse lesion in the dorsal region, with good recovery of sensibility and some recovery of power in the legs. There is considerable rigidity of the legs, and phasic flexor spasms are invariably present, and there is more sphincter trouble present than the condition of recovery of motion and sensation would lead one to expect. There are three kinds of pain: Dull persistent pain, sharp lancinating pains, and cramp. They are said always to disappear if the patient recovers. His experience is that they certainly persist if he does not recover.

There are three possible explanations of these pains: (1) That they may be caused by irritation of the sensory tracts in the region of the lesion; (2) that they may be caused by meningeal adhesions and matting of the cauda equina by the organization of intrathecal extravasation of blood; (3) that they may be the result of the muscular rigidity and flexor spasms. Col. Gordon Holmes is in support of the first of these causes, since in some cases the pain is distributed from the lesion downward to the feet. The similarity of these pains to those which obtain in conditions of chronic spinal meningitis suggests that meningeal adhesions and irritation of the roots are present in some of the cases. The fact that they disappear as recovery advances and persist if there is no further recovery points to the condition of local muscular spasm as being the chief cause in some of the cases.

RARE SYMPTOMS.

Among the rare symptoms which have been described, hypothermia and anuria in cases of cervical lesion and

vomiting in mid-dorsal lesions have not come under my observation. These are early symptoms and are either rapidly fatal or they do not persist, so that we do not see them in the later stages. Pyrexia and tachycardia in cervical cases have both come under my notice, as has also polyuria in dorsal cases. Herpes zoster from injury to the root ganglia is necessarily an early symptom and has not occurred in any of Collier's cases. Neither has he observed anything approaching the remarkable rhythmic movements which have occurred early in so large a proportion of Col. Holmes's cases of lesions in the upper lumbar region.

Cervical shivering has been met with in lesions of the lower cervical and the upper three dorsal segments only, and the lesions in all the cases have been severe.

PROGNOSTIC INDICATIONS.

In nearly all the cases the initial clinical picture is that of a total lesion of the cord, and in the early stages for prognostic indications as to how much of the transverse lesion is anatomical and how much physiological we must look to the condition of the reflexes. The early reappearance of the plantar reflex if lost at first and the change from the flexor to the extensor type soon after the seventh day, or the presence of an extensor response earlier than this, are certain indications that the lesion is partial and that some recovery will occur. Persistent loss of the plantar reflex and long-lasting flexor response are indications that the lesion is severe and that useful recovery is highly improbable. Early return of the knee jerk and of the ankle jerk is of good prognostic import. Even in the less severe cases the knee and ankle jerks do not return before the fourth day, and their reappearance before this date gives hope that complete recovery may occur. Col. Gordon Holmes is of opinion that the presence of an extensor response is a certain indication that the lesion is incomplete. These remarks apply to each leg individually. The condition of the reflexes on one side has no bearing as to the recovery of the opposite limb. The further prognostic indications are simple. They depend upon the date of return and the rapidity of return of sphincter control, of sensibility, and of motor power. Early return of sphincter control is one of the best of signs. In two cases where recovery has been complete, and in two other cases where recovery has been nearly complete, some sphincter control returned within a week of the injury.

It is only natural to suppose, in spite of the clear exposition by Holmes, that a legitimate difference of opinion should exist regarding the institution of operative treatment. These cases are hopeless in such a large proportion of instances that the desire to aid them by operative measures is a most human impulse. In the following

abstract of the Congress at Königsberg, Michaelis adheres to conservatism, whereas Guleke finds himself forced into a more radical position. Marburg and Ranzi lend unqualified approval to the doctrine of conservatism. Leva emphasizes the important point that spinal shock may early simulate complete severance of the cord.

Michaelis: Injuries of the Spinal Cord (Verletzungen des Rückenmarks *International Abstract of Surgery*, Mar. 1916.)

Michaelis, at a meeting of the *Verein für wissenschaftliche Heilkunde*, of Königsberg, reported the case of a patient wounded by a bullet which inflicted a small wound of entry in the back of the neck, somewhat to the left of the middle line, and at the level of the fourth cervical vertebra. There was no wound of exit, though the lower jaw on the left side was badly shattered. A day after the infliction of the wound both arms and legs were paralyzed, but there was no paralysis of the bladder. The paralysis of the right arm and leg soon began to disappear, leaving only a slight weakness of the right arm. A skiagram showed slight injury to the fourth cervical vertebra at the junction of its body with its arch. Viewed from in front, the bullet could be seen lying behind the much shattered horizontal ramus of the left lower jaw. No active treatment was attempted during the first fortnight, as it was hoped that the paralysis of the left arm and leg would disappear spontaneously. Treatment of the fractured vertebra by Glisson's extension apparatus was impracticable, owing to the fracture of the jaw. The fragments were united by bronze wires and the bullet was removed. As there was no improvement in the paralysis of the left side after a month, laminectomy was performed, on the assumption that the paralysis was due to pressure on the cord by a fragment of bone or to peripachymeningitis with local edema of the cord. The arches of the third, fourth, and fifth cervical vertebrae were removed, but no loose fragment of bone could be found. There were, however, signs of peripachymeningitis. To the left of the middle line the dura was adherent to the fourth cervical vertebra over a small area. This adhesion was severed. After the dura had been opened and a considerable quantity of cerebrospinal fluid had escaped, a narrow groove was seen passing across the cord from behind and to the left, forward and outward. This wound of the cord, which had been gouged by the bullet, was closed by catgut ligatures passing upward and downward so as to unite the upper and lower margins of the groove. The wound in the dura was then closed and the operation completed. Three days later the movements of the left big toe were regained, and during the following days the paralysis of the left leg gradually receded upward. Eleven days after the operation there were active movements about the ankle, and three days later the patient could also move his legs

slightly about the knee. Movements of the left arm did not begin to return till about four weeks after the operation, when first the thumb, then the fingers, and finally the rest of the arm began to regain the power of movement, the paralysis gradually receding upward. Though the use of the left leg was almost completely restored, that of the arm remained much impaired. Michaelis, while insisting that the operation was very successful, admitted that the reason for this success was not perfectly clear. It might have been due to relief of pressure on the cord, which, in its turn, might be traced to the drainage of cerebrospinal fluid. The improvement might also have been largely due to the closing of the groove in the cord.

Leva reports that he has often seen paralysis of the bladder and intestine, as well as sensory disturbances, disappear spontaneously, and marked improvement in other symptoms occur, even in cases in which there was evidence of complete transverse section of the cord (*vollständige Querschnittläsion*). It was therefore unwise early in the case to diagnose total division of the cord and to give an unfavorable prognosis. Brown-Séquard's unilateral lesion did not run such a favorable course as the total transverse lesion (*Querläsion*). In some cases of injury to the cord there were only a few isolated symptoms. Thus, in one case in which certain nuclei of the medulla were involved, the symptoms were paralysis of the recurrent nerve and unilateral atrophy of the tongue. In another case the symptoms consisted of difficulty in swallowing, loss of the patellar reflexes, and static-atactic manifestations. In a third case weakness of the legs, diminution of the tendon reflexes, and diffuse sensory disturbances were observed.

Guleke said that he had come to the conclusion that it was often extremely difficult to learn the extent to which the cord had been injured, and that he was therefore in favor of early operation as a rule, for, though this principle led to superfluous operations, it also saved the lives of many who would otherwise have died. He had performed 20 laminectomies, and in none had any harm been done. He did not advise this operation in cases complicated by severe pneumonia, meningitis, open and much infected wounds, or "urosepsis." Hemothorax, on the other hand, is no contra-indication, but when it is present the operation should be performed under local anesthesia. In 10 of his cases the cord was completely crushed, and they all terminated fatally; in five other cases death was due to sepsis. There were, therefore, only five recoveries among his 20 cases; but he was certain that three of the patients who recovered would have died had not the operation been performed. In these three cases splinters of bone or bullets were found in the cord, which they had much injured.

Marburg, O., and Ranzi, E.: Spinal-Cord Injuries Due to Bullets (Über Rückenmarkschüsse). *Wein. klin. Wchnschr.*, 1915, xxviii, 113.

The authors report a series of 35 spinal-cord injuries treated at the von Eiselsberg Clinic, Vienna. Although nothing particularly new is offered, the conclusions drawn may be summarized as follows:

1. In contradistinction to brain injuries it is essential to wait a considerable time (four or five weeks) until the condition has become stationary before a laminectomy is performed.

2. The operation is contraindicated in the presence of pulmonary or abdominal complications; likewise if severe suppurative processes or decubitus is present near the site of operation; also if the case is complicated by a suppurative ascending pyelitis.

3. Mild infection of the urinary tract and granulating bed sores and not contraindications.

4. In spite of the small clinical material presented it is evident that severe direct injuries and tangential shots, in contradistinction to indirect injuries, such as compression, edema, liquor stasis, and local inflammation, are hardly adapted to radical surgical intervention.

Leva, J.: Injuries of the Spinal Cord in War (Über Verletzungen des Rückenmarks im Kriege). *München. med. Wchnschr.*, 1915, lxii, 925.

In 9 of the cases observed by Leva symptoms of complete transverse lesion predominated, in 5 there were signs of a lesion of one-half of the cord, and in 7 signs that indicated solitary injuries of different centers. Of the patients with signs of a total transverse lesion 2 died soon after they were received, 1 of an ascending meningitis and 1 of pyelonephritis; the condition of 1 remained unchanged, in 1 the symptoms gradually improved till they were those of a unilateral lesion, in 2 a spastic paralysis developed, and 3 improved so much that no organic symptoms remained except increased reflexes.

These cases show that symptoms of a complete transverse lesion at first do not by any means prove that there is actually a complete severing of the cord. Shock and concussion have caused a temporary cessation of its function, which is restored in time.

Of the 5 patients with unilateral symptoms 2 improved markedly, while the paralysis only improved slightly in the other 2. In the other cases the course varied; in 1 it was very favorable, 2 developed cerebellar symptoms, 1 developed pain and atrophy of the right arm, which did not improve much even after laminectomy. In 2 cases of injury of the neck signs of lesion of the medulla developed, including various subjective symptoms and paresthesias. Injuries of the cervical column did not produce such serious effects as

injuries lower down. The total of 21 cases after six months' observation shows 2 deaths, or 9.5 per cent, but they can not be regarded as closed, for secondary signs of degeneration often appear a long time after the injury.

Perthes, Goldstein, Armour, Guillain, and Hull all incline to a somewhat radical position. It is interesting, however, to note the strong tendency to qualify their statements, thus mirroring the uncertainty with which one approaches these grave lesions.

Perthes, G.: Laminectomy in Cases with Bullets Lodged in the Spinal Cord. (Über Laminektomie bei Steckschüssen des Rückenmarkes). *Beitr. z. klin. Chir.*, 1915, xcvi, 76.

There is still a great difference of opinion as to the proper course to pursue in gunshot injuries of the spinal cord; some surgeons advise operation, and others equally skilled advise against it. Perthes considers only those cases in which the projectiles remain in the spinal canal, and gives the histories of six such cases operated upon by him. Two of these patients died the day after the operation; one died later, after the wound had healed; one recovered from the operation, but not from the paralysis; but in the two other cases the improvement after the operation was so marked that there is every reason to believe it will be complete.

He discusses the symptoms of complete and partial transverse section of the spinal cord and concludes that laminectomy should be performed in all cases where there is only partial section. In such cases the symptoms are often due to pressure by the projectile, and recovery after operation is remarkably rapid and complete. *If there is complete transverse section of the cord, operation is useless, but it must be borne in mind that there are often clinical signs of complete section when anatomically a part of the cord is preserved;* so it is quite possible that some such cases may be saved; at any rate the operation can do no harm, for the patients will die if not operated upon. The operation should be performed under local anesthesia with the aid of pantopon-scopolamine or scopolamine-morphine anesthesia. *In the cases of only partial section of the cord the operation should be performed at once;* there is no object in waiting, as the pressure symptoms will only grow worse.

Goldstein: Gunshot Injuries of the Brain and Spinal Cord (Beobachtungen an Schussverletzungen des Gehirns und Rückenmarks). *Deutsche med. Wchnschr.*, 1915, xli, 215, 250.

There are three groups of such injuries: (1) Those that are so severely injured that they die soon afterwards; (2) those in which the symptoms are very severe at first, but improve in a relatively short time and after a few weeks almost disappear; (3) those in which the symptoms do not improve, and in spite of the best care the patients die after

a few weeks. Of course only the latter two classes are seen in the hospitals.

In injuries of the spinal cord he advises more frequent operation. He describes two cases in which autopsy showed that operation might have been useful. In one there were bone splinters in the cord that might have been removed, and in the other connective-tissue adhesions that might have been freed to relieve the cord from compression.

He advises operation in all cases where there are evidences of a transverse lesion and where flaccid paralysis with failure of reflexes persists for some time. The length of time before operation depends in part on the patient's general condition. If this is bad and there are marked bladder disturbances and severe decubitus, not more than three weeks at the most should elapse. Of course operation may be in vain if the cord is completely severed, and there is no way of telling absolutely from the clinical symptoms whether this is true; but the prognosis is hopeless in these cases anyway and no harm can be done, whereas by operating cases will be saved in which there is any possibility of cure. *Operation should always be performed in cases where a bullet can be seen in the spinal canal in the röntgen picture and the disturbances do not improve.*

Armour, D.: Gunshot Wounds of the Spine; Their Surgical Aspect.
Lancet, Lond., 1916, cxc, 770.

The author divides gunshot wounds of the spine into two classes: (1) Those in which there is no interference with the function of the spinal cord; (2) those in which there is more or less interference with the function of the spinal cord with or without obvious injury to the vertebral column.

Injury to the vertebral column may be followed later by effects of inflammatory products—adhesions, narrowing of the spinal canal, intra or extra dural clot, etc.

Immediate injury to the cord may be caused by: (1) The missile passing through part or the whole of the cord; (2) fractured bone causing compression, contusion, laceration, or complete division; (3) concussion.

The author has found the X ray to have only confirmatory value in localizing bone injury and foreign-body position.

The points of importance arising regarding operative intervention are: (1) Will any benefit to the patient result from the operation? (2) Will his life be endangered by the operation? (3) Will he be made worse by operation?

The author then discusses indications for operation and urges interference under proper surgical skill and asepsis in all cases in which complete section has not taken place, providing the patient is in a fair general condition. He says: *"It is unfair to the patient and unfair to surgery to wait on and on till hope gives place to despair and then call in a surgeon as a last resort to perform the impossible."*

Operation is therefore indicated (1) to relieve pressure from depressed or displaced fragments of bone; (2) to relieve pressure from blood clot or from extensive hemorrhage, either extra or intra dural; (3) to relieve pressure and prevent further destruction from edema by enlarging the constricted bony canal; (4) to remove the danger of pressure from exudate and inflammatory thickening.

Guillain, G., and Barré, I. A.: *Injuries of the Spinal Cord in War* (Les plaies de la moelle épinière par blessures de guerre). *Presse méd.*, 1916, p. 497.

In the present war injuries of the cord are most frequently due to shell fire. Of the authors' cases 61 per cent were due to shells, 23 per cent to bullets, 8 per cent to shrapnel.

In addition to the usual symptomatology, paraplegia, disturbance of muscular tonus, neuromuscular contractility, abolition of reflexes, etc., the authors have observed in paraplegic patients with abolition of all tendon reflexes, that after percussion of the rotulian tendon even with the quadriceps muscle remaining absolutely inert, there is a more or less vivid contraction of the posterior thigh muscles, most frequently of the postero-externo muscles with a sometimes slight flexion movement of the limb which gives the appearance of what has been termed inversion of the rotulian reflex. This is a true reflex and may be aptly termed the posterior tibiofemoral reflex.

While sensory painful disturbances are lacking in the majority of cord injuries, tactile painful anesthesia is most frequently absolute and total. Muscular atrophy is sometimes extremely rapid; urinary and fecal retentions are almost always present.

In the authors' opinion a description of the general symptomatology in injuries of the cord is a chapter still to be written, as it is not to be found in any text on neurology. In the beginning for the first few days the patient feels relatively well and has no appearance of severe injury. The two most marked symptoms at this time are excessive thirst and insomnia. Sooner or later the appetite which was good is lost, loss of weight is rapid, and somnolency is almost constant.

Of 100 cases in the authors' service the mortality was 80 per cent. Of the other 20 evacuated cases several are known to have since died, and the authors are in reality only cognizant of 4 cases of amelioration, 2 subsequent to surgical intervention and 2 spontaneously.

In a table given by the authors it is seen that most cases do not survive three weeks. The maximum survival observed 57 days in a case of lesion of the eighth dorsal segment.

What are the real causes of such rapid death in these injuries of the spinal cord? The authors believe that urinary and pulmonary infections which have been indicated by some as the cause may be excluded. Patients injured in the sacral or dorsal regions usually succumb to a purulent meningitis,

but the principal cause appears to be a progressive cachexia. The causes of death according to the authors' view should be classed as: Purulent meningitis; disturbance of the sympathetic nervous system of the digestive tract, abdominal viscera; and vascular glands; cachexia through default of assimilation; anemia of the cerebral centers. If the lesion is very grave, the sympathetic nerve trouble is at a maximum.

There is little difficulty in diagnosis. *The only question is one of differentiation between complete and incomplete section, or a hematomyelia, a medullary disturbance, or a compression.*

In complete section (anatomic or physiologic) motor paraplegia is complete; urinary retention absolute; all the tactile painful, thermic, and vibratory reflexes are abolished; all the tendon reflexes are abolished. In incomplete section the abolition of sensations (especially vibratory) is not absolute nor global, even segmental attitudes may be preserved. Traumatic hematomyelia is almost always accompanied by a sanguinary suffusion in the piamater-arachnoidean space which may be demonstrated by lumbar puncture.

The authors think that in all spinal injuries radiography is indispensable not only to show the nature of the osseous lesions, but also to determine the rachidian or extrarachidian situation of the projectile.

Treatment consists in the association of neurology and surgery. *Every spinal wound should be explored as quickly as possible*, the entry orifice stripped, the wound disinfected, and the bone examined. All fragments should be removed. The authors discountenance the use of antiseptics which may be hurtful to the exposed medullary tissues. Manipulation in this region should be as delicate as possible. Chloroform or ether as anesthetics are very badly supported and the authors prefer a local anesthetic.

If on a prior examination there is no evidence that the dura mater is opened, the absolute rule of surgery not to open it must be respected. But if it is open prolonged lavage with warm physiologic serum at slight pressure is the only treatment. Any attempts at suturing according to the authors' experience is absolutely useless.

The question of removal of the projectile is open to discussion. If it is situated at the back or at the sides of the cord or if it is intramedullary, it should be removed. When the projectile has traversed the cord causing perhaps only a partial section and is lodged in a vertebral appendage its removal although possible from the surgical viewpoint is a matter of opinion, because in such event new lesions will be created which may turn an incomplete section into a complete one.

Frangenheim, P.: Gunshot Injuries of the Spinal Column and Spinal Cord (Schussverletzungen des Rueckenmarks und der Wirbelsaule). *Muenchen. med. Wchnschr.*, 1915, lxii, 1473.

Frangenheim has operated in 25 cases; of these, 9 have died and the others were discharged after four to six weeks'

treatment, some of them improved, and some, in whom there was only partial paralysis, practically well. There is no way of making a certain diagnosis as to the extent of a spinal injury. It is very difficult to localize projectiles accurately. Fractures of the spinous processes are hard to demonstrate roentgenologically, and small fragments broken off from the bone and compression of the cord from bone fragments can not be so demonstrated. As a rule splintering of the bone can not be demonstrated till the muscles have been stripped off from the processes.

Contusion of the cord, compression of the cord, and complete transverse severing of it all give the clinical picture of complete paralysis with loss of control of the bladder and rectum. It can not be determined from the reflexes whether the cord is completely severed or not; therefore the author advises exploratory laminectomy in all cases; further treatment depends on the findings. Early operation is generally preferred to expectant treatment.

Lumbar puncture over the site of the injury is of considerable value in diagnosis. Frangenheim admits that his numbers are small and that later complications may yet appear in some cases, but he is convinced that exploratory laminectomy is the best method of procedure in these cases.

Hull, A. J.: *Treatment of Gunshot Wounds of the Spine.* *Brit. M. J.*, 1916, 1, 577.

To be successful spinal operations must be performed at an early stage before any vital changes have occurred in the cord. *By delaying operative interference cases lose their chance of recovery either by the sepsis spreading or by pressure on the nerve tissue, causing these vital changes to take place.*

It would appear justifiable to operate upon spinal injuries when the X-ray localization shows a foreign body present in an accessible position, and especially when there is evidence of some remaining conductivity of the cord, as here the removal of pressure may be followed by great improvement. Pain in some spinal lesions is so intense that an operation is justifiable, whatever the lesion of the cord.

Three lines of treatment are indicated: (1) Prevention of sepsis, (2) removal of gross pressure upon the spine, and (3) the prevention of complications which threaten life.

Marie and Roussy draw attention to the important question of after care of spinal-injury cases, furnishing specific details for the avoidance of decubitus. Under this head it should be noted that verbal reports have reached this country that in many of the hospitals abroad the spinal-injury cases are not catheterized or irrigated. The bladder is allowed to empty itself by overflow.

Marie, P., and Roussy, G.: Possibility of Preventing Decubitus in Wounds of the Spinal Cord (Sur la possibilité de prévenir la formation des escarres dans les traumatismes de la moelle épinière par blessures de guerre). *Bull. Acad. de méd., Par.*, 1915, lxxiii, 609.

Though the prognosis in injuries of the spinal cord is grave, it is by no means so hopeless as it has usually been considered. Paraplegias often show a remarkable tendency to spontaneous recovery. On account of the feeling of hopelessness in these cases precautions have been neglected that might have improved the condition of the patients.

It has always been held that decubitus was caused directly by the injury of the spinal cord itself, and that therefore it could not be prevented. This is untrue, and bedsores can and should be prevented in all cases. The patient can not change his position on account of the paraplegia, so that the same parts have to support the weight of his body constantly. Prolonged compression interferes with the circulation in these parts. Moreover, because of the loss of sensation the patient does not have the normal inclination to change his position. These factors, however, only produce a dry eschar that is not at all serious, but because of the lack of continence they become soaked with urine and then infected. That this is the cause, and not the spinal injury, is shown by the fact that the site of the decubitus has no relation to the level of the cord injury. Wherever the cord injury may be, the bedsore occurs at the points of pressure on the sacrum.

To prevent the formation of these sores the bladder and rectum should be examined in every case of injury of the spinal cord. To avoid soiling with urine a retention catheter should be inserted. The bowels may be locked for a few days by the administration of opium, and the skin may be protected with talcum powder or vaseline. The patient may be placed on air cushions while being transported. If he has been neglected during transportation and arrives at the base hospital with bedsores already developed, they may be cured if he is given the greatest care and the sores dressed once or twice a day with phenolized powders. Nurses should be instructed to change the patient's position every hour during the day and every two hours at night. Infections of the bladder and urethra should be treated with irrigations of potassium permanganate or nitrate of silver.

The two following abstracts by von Eiselsberg and Ascher & Lichen represent the latest authoritative German opinion on gunshot injuries of the spine. These two abstracts are grouped separately merely by reason of their late date.

Von Eiselsberg: Report on Gunshot Injuries of the Spinal Cord. (Ueber Schüsse insbesondere Spaetchirurgie.) Report to the Second German Surgical Congress. *Beitraege zur. Klin. Chir. Bd. v. Hft 1. Kriegschirurgische Hefte.* 1916.

The problem of transportation of fresh injuries of the spinal cord is most important. In many cases it is desirable, if possible, that the wounded lie on the stretcher upon which they are first placed until they reach their ultimate destination. This is doubly true with patients suffering from spinal cord injury. Not only that, but they should be fixed to the bar in order to avoid alteration of position and in order to guard against jolting. Special spiral springs should be put under the legs of the stretcher and, best of all, the kind of spring used which can be screwed to the floor of the carriage.

Perthes has warmly recommended early operation for spinal cord injury. This does not correspond with the opinion of von Eiselsberg, who operates early only in very exceptional cases. He says, further, that it is usually not possible to operate until the patients reach the base hospitals.

Speaking of the necessity for catheterization, it is even more true in war than in peace time that it is practically impossible to keep the bladder free from bacteria; particularly during transportation is this true. In order to avoid danger of infection through repeated catheterization or permanent catheter, von Eiselsberg recommends a suprapubic cystostomy.

As soon as the patient reaches the base hospital, it is most important of all to determine the location and the extent of the spinal cord injury. In cases where one recognizes a fracture and where one finds the projectile in the spinal canal, he should do an early operation, for the reason that a long-continued pressure on the spinal cord can produce a second permanent degeneration. A second indication for an early operation is a severe infection with high fever and pus discharge.

A very important part of the treatment lies in protecting the patient against the development of bedsores. These may develop very early and very rapidly, even during the transportation from the field.

Injuries of the spinal cord call for two classifications. The first classification is according to anatomical injury; the second according to the symptoms. The pathological anatomy teaches that several injuries may be produced:

1. Hemorrhage into the dural sheath, and through this compression symptoms. Hemorrhages may be completely absorbed. When not completely absorbed they organize, and this may result in pachymeningitis, or the so-called "meningitis serosa circumscripta." The regression of a traumatic hematoma is quite analogous to the traumatic cysts seen over the brain cortex.

2. Projectile or bone fragments may cause pressure on the spinal cord.

3. At the time of injury an irreparable damage may have been done to the spinal cord.

Obviously, combinations of these three findings usually occur, and the paralysis may be caused partially by a hematoma, partially by an impinging vertebra or fragment of bullet, and partially by a cutting of the spinal cord by bone fragment or projectile. The diagnosis upon anatomic considerations is usually purely conjectural.

According to the symptomatology there are three groups, one with complete transverse lesions, in which there is absolute flaccid paralysis of the parts below the site of spinal-cord injury, with loss of reflexes and complete disturbance of sensation. This picture of complete lesion can be caused by compression, either from vertebra or projectile. Second, there is spastic paraplegia where there is paralysis with increased reflexes, clonus, positive Babinski, and this is regarded generally as compression syndrome. In these cases laminectomy promises a better outlook. A third group of partial spinal-cord lesions is divided into two forms: (a) The Brown-Séquard type and (b) the spinal hemiplegia.

4. Lesions of the cauda. Here the prognosis is good, except for the bladder troubles.

It often happens that there is complete loss of control of the bladder when the injury of the spinal cord is not complete, and the kind and the location of the spinal-cord lesion seems to bear no relationship to the kind and the duration and the prognosis of bladder disturbances. The chief danger in these bladder disturbances is the bladder and kidney infection. The danger of ascending infection is the gravest of all dangers in spinal-cord injuries, except those where the injury is above the phrenic nerve.

Now comes the grave question for the surgeon to decide—whether and when operation shall be done. *The symptoms are often confusing and differential diagnosis between the kind of injuries can not be made, particularly since combinations often occur; but in order to avoid operating upon cases which would improve without operation, von Eiselsberg has made a practice not to operate until 8 or 10 weeks after the injury, meanwhile observing symptoms most carefully. When there is the slightest improvement he postpones operation and operates only when the condition remains stationary or where symptoms grow more pronounced. As before mentioned, he makes exception in those cases where impinging bone fragments or projectiles can be recognized.*

Ether is the anesthetic of choice, and the important points in technic are care to avoid producing further injury and very, very wide exposure, and opening of the dural sac. The treatment before as well as after the operation demands the most careful and conscientious nursing. Patients should be kept on water cushions or water mattresses. The water mattress is very pleasant to the patient, and by its use bedsores can be avoided. Very often from large bedsores infection spreads and a certain cachexia develops which complicates the picture. It is also of very much im-

portance from the beginning to avoid atrophy and contractions by massage, electricity, and careful passive movement.

Forty cases of spinal-cord injury were operated. Of these, 16 were distinctly improved, 8 were slightly improved, 7 were unimproved, 2 died soon after the operation, and 7 died eventually. In all, there were 9 deaths and 24 improved cases out of 40 operative cases. In 73 cases not operated there were 36 deaths and 35 cases showing improvement. This shows much better results in the operative cases, in spite of the fact that the operated cases were more severe and were cases in which no spontaneous improvement was noted.

Ascher and Licen: Gunshot Wounds of the Spine and their Treatment (Über Schussverletzungen des Rückenmarks und deren Operative Behandlung). *Beiträge zur Klinische Chirurgie. (Kriegschirurgisches Heft XXXIII.)* Mar., 1917. P. 521.

Ascher and Licen report 35 cases of gunshot of the spinal cord, which they treated for several months. Twenty of the 35 cases were through and through; in 15 cases the bullet remained in the body. In only 1 case was the bullet free in the spinal canal. The mortality was 25 per cent.

The diagnosis of spinal-cord injuries has three points to consider—the location of the lesion, the degree of the lesion, and the kind of lesion. In localizing the injury the path of the bullet, as estimated by the entrance and exit wounds, gives a valuable clue, although it should be remembered that a projectile does not always traverse a straight line, but may be deflected by the spinal column, and also that the wounds often lie in a movable region, as, for instance, the shoulder, and that it is impossible to tell in what position the patient was at the moment of his injury. Deformities in the spinal column did not help in localizing the lesion in these cases, and while sensitiveness over the spinal processes was present it extended also to uninjured vertebræ. Crepitation was never encountered.

X-ray examinations were in large majority of cases unsatisfactory. The neurological examination is to be most relied upon in establishing the exact location of injury. It is to be remarked that the neurological findings indicated an injury somewhat higher than actually found, ordinarily two or three segments of the cord. This is not only during the first days, but also later and possibly permanently so.

The degree of the lesion can usually be determined within the first few days, certainly within the first two or three weeks, and chiefly through exact and oft-repeated examinations. The kind of lesion can seldom be determined upon.

Spinal puncture did not help these cases. In discussing operative treatment, in 12 of the 35 cases operation was considered indicated. In 1 of the 12 operations there was complete recovery. In 1 there was very marked improvement; 4 were uninfluenced by the operation; 3 improved very slowly after the operation, although at operation no

spinal-cord injury was found, and it was questionable whether improvement was due to the operation; 3 cases died within the first 10 days after laminectomy.

Laminectomy was performed under local anesthesia, with the exception of one case. The wound was closed tightly. No case of meningitis developed. Primary healing occurred in each instance.

Operation should be undertaken at the very earliest opportunity in all cases in which there is compression of the spinal cord, without consideration of the severity of symptoms. Operation should also be undertaken in those cases in which X-ray examination shows a possibility of bone splinters lying within the spinal canal and in those cases in which there is a sudden increase of paralysis. In the vast majority of cases in which nervous examination shows a complete lesion of the cord operation is superfluous. Other authorities are quoted who recommend operation only when there is no improvement after a few weeks' observation.

CHAPTER III.

PERIPHERAL NERVES.

PART 1.

DISEASES OF THE PERIPHERAL NERVES.

By GORDON M. HOLMES, M. D.

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The symptoms of disease of the peripheral nerves may be classified as they result from disturbance of the functions of those fibers which convey impulses to the muscles, of those which carry the sensory impressions centralward, and, finally, of the fibers which are more immediately concerned with the nutrition of the tissues, either directly or as part of the mechanism for the local regulation of the blood stream. There are, however, but few nerves in the body, excepting the cranial nerves, which are either entirely sensory or entirely motor. The consequence is that when any nerve is severely injured, both sensory and motor symptoms and, probably, in addition, nutritive or trophic changes result. It is by the extent and distribution of these symptoms that the localization of the disease can be determined. If, however, the lesion or the disease of a mixed nerve is partial or incomplete, the functions of the different sets of fibers may suffer unequally; the general experience is that in incomplete lesions, as those due to compression, the sensory suffer much less than the motor fibers, or sensory symptoms may be absent, although the muscles supplied by the nerve are completely paralyzed. Luderitz showed experimentally that the conductivity of the motor fibers is lost earlier than that of the sensory fibers when a mixed nerve is subjected to slowly increasing pressure. Evidence of trophic or vasomotor disturbance is, as a rule, little apparent in partial lesions.

Motor symptoms.—The symptoms which immediately follow the complete interruption of the motor nerve fibers that supply a muscle are complete paralysis of both reflex and volitional contraction of that muscle, loss of its tone, and, later, atrophy, changes in the character of its response to electrical stimulation; and, finally, if recovery does not take place, contracture, owing to secondary fibrosis. These features distinguish lower motor neurone or spino-muscular

paralysis from paralysis due to disease in the upper motor or cerebro-spinal neurones. In the latter condition movements, not muscles as such, are paralyzed, and the distribution of one or more peripheral nerves; secondly, the tone of the paralyzed muscles is increased, not diminished or lost; that is, the paralysis is spastic and not flaccid, and the reflexes which are dependent on the tone of the muscle, as the knee jerk, are more active than normal instead of being abolished; thirdly, the paralyzed muscles do not atrophy, or atrophy only to a much less degree; and, finally, there is no change in the nature of their response to electrical stimulation.

The amount of loss of power in the muscles naturally depends on the degree of the injury of the nerve; when there is complete interruption of its structure or loss of its function the paralysis necessarily complete. When the lesion of the nerve is incomplete another factor must be considered, namely, the rate of its evolution, as when the disease is of sudden or rapid onset the symptoms may be considerably greater, for a time at least, than those produced by a similar lesion which has developed slowly. A muscle that receives its motor fibers from more than one nerve is not completely paralyzed by even a complete lesion of one of them.

The normal tone or tension of muscles is dependent on the integrity of the peripheral reflex arc, which consists of the afferent fibers from the muscle that enter the cord by the dorsal spinal roots and terminate by synapsis around the cells of the corresponding motor neurones in the ventral horn, and of the peripheral motor neurones. When this arc is broken in any place the muscles immediately lose their tone. The atonia may be recognized by loss of the normal contour of the muscles, if this is easily visible; by their softness and flabbiness to touch, and by the lack or diminution of the resistance which they normally offer to stretching, and consequently the excess of mobility to passive movement of the joint at which they act.

The deep reflexes or tendon jerks are dependent on the maintenance of the muscle tone, and it is by these so-called reflexes that this variety of tone in the muscles is most easily measured. Their disappearance or abolition is consequently an indication of the diminution or disappearance of the tone of the muscles concerned and not of their paralysis alone. The knee jerks may disappear early in the toxic affections of the peripheral nerves before there is any demonstrable evidence of either motor or sensory paralysis; and during recovery from such conditions, or after regeneration of degenerated nerves, they may be absent when the recovery of power is apparently complete. The diminution or loss of tone, as measured by the deep reflexes, is the most sensitive guide to any interference with the normal function of the peripheral nervous system.

Atrophy of the muscles supplied by the affected motor fibers is one of the most prominent symptoms; this is due to degeneration or regression of the muscle fibers owing to loss of the trophic influence which the motor nerves normally exert on them. It is recognizable by a diminution in the size of the muscle and by its soft and structureless consistence. The latter symptom is frequently neglected, but it may prove valuable, especially in children, in whom it is not easy to determine the distribution of an atrophic paralysis by merely noting the movements which can not be performed; here by touch alone we may pick out the affected muscles. Muscular atrophy is generally recognizable within two or three weeks after a complete interruption of a motor nerve, and increases rapidly from this time until few if any fibers remain. When the nerve lesion is incomplete, the atrophy corresponds closely with the degree of paralysis; in other words, the conducting power and trophic function of the peripheral motor nerves are lost together. When the lesion of the nerve is very slowly progressive the muscular atrophy is generally less prominent than the paralysis.

Coincident with the atrophy of the fibers of the wasting muscle an increase and proliferation of its connective tissue occur. If the muscular atrophy attains severe degrees, and if regeneration does not soon set in, this new connective tissue slowly undergoes fibrosis and the muscle is converted into a firm, inelastic band. The contracting fibrous tissue may produce contractures and deformities. Further, for the recovery of function it is necessary that the muscle fibers should regenerate, and regeneration must be evidently seriously interfered with by the presence of dense and contracted fibrotic tissue. The condition of the muscles and the changes they undergo are therefore as important as the changes in the affected nerves.

One of the most valuable signs in disease of a peripheral motor nerve is changes in the electrical excitability of the nerve and of the muscles it supplied. Muscular contraction can be normally obtained by either faradic or galvanic stimulation of the motor nerve fibers which supply it. Two or three days after section of a nerve the excitability of its peripheral portion is diminished, and after a period of six to eight days the excitability is lost with the secondary degeneration of the nerve. The most characteristic changes are observed on direct stimulation of the muscle; they compose what is known as the reaction of degeneration (R. D.) and usually appear from the eighth or tenth day after section of the nerve. It must be mentioned that according to Sherren increased excitability to the galvanic current is found only when the lesion of the nerve is incomplete. After some months, if recovery has not set in, the reaction of the muscles to the galvanic current diminishes slowly and finally disappears.

In every case of peripheral paralysis it is important to test the electrical excitability of every portion of the nerve and muscle that can be reached. It occasionally happens when there is a local lesion in the nerve that muscular contractions can be obtained by stimulation below the lesion, but not from above it; the lesion may block the conduction of impulses without leading to secondary degeneration of the peripheral portion of the fiber. On the other hand, regenerating nerves may be excitable only from above the lesion; the regenerated portion is for a time inexcitable to stimuli which it can conduct. This may depend on the absence of a myelin sheath (Erb). When the lesion is unilateral the excitability of both nerve and muscle may be compared with those of the normal side; when both sides are paralyzed the irritability should be compared with those of a normal person, or the tables prepared by Stintzing to show the normal limits of excitability may be used.

Ghilarducci's distal reaction is also a valuable test; the indifferent electrode is placed on the neck or back, the other distal to the insertion of the degenerated muscle; then on closure of the current a contraction of this is obtained by a weaker current than suffices for the unaffected muscles.

Symptoms of irritation of motor fibers are much less frequently met with in diseases of the peripheral than of the central nervous system. Spasm, either tonic or clonic, and cramps, when due to irritation of the peripheral neurones, are generally reflex in origin and arise from excitation of the sensory fibers. Occasionally, however, intermittent or tonic spasm may result from irritation of a motor nerve by a neighboring focus of inflammation or by a foreign body, but even here it is difficult to exclude its reflex origin. True reflex spasms are much more frequent; the involuntary facial movements which often accompany trigeminal neuralgia, and even irritation of the cornea, are of this nature. The fixation of painful joints by the tonic contraction of the surrounding muscles is also due to irritation of the sensory fibers which reflexly excite the corresponding motor neurones to excessive tonic activity; the sensory impulses may, if sufficiently intense, spread to wider reflex centers in the cord. The potency of sensory impulses from the periphery in the causation of spasm is illustrated by the severe spasms met with in strychnine poisoning and in tetanus; both these poisons only transmute an inhibitory effect into an excitation effect in the spinal reflex centers (Sherrington), and the spasms or convulsions are always directly due to a stimulus from the periphery. Cramp of a muscle may be occasionally due to influences that affect its fibers directly, as poisons like veratrine; vascosity of the blood may also predispose to or cause spasms.

Some forms of muscular atrophy are characterized by the occurrence of fibrillation or intermittent, more or less rhythmical wavelike contractions of some of the fibers of a muscle; this is more probably due to direct stimulation of the fibers than to excitation of the motor nerves. Myokymia, a condition characterized by constant undulating or wavelike contractions of the muscle fiber which changes from place to place, has been regarded as a symptom of abortive neuritis, but is rarely seen.

After complete division of a nerve and primary suture the time necessary for the return of power depends on the distance of the lesion from the periphery; Sherren lays down the rule for the upper extremities, that when a nerve is divided at the wrist perfect power may be regained within a year; but if at the elbow or in the plexus not for two years. The muscles nearer the lesion regain their functions earlier than those distant from it. Recovery is slower after secondary than after primary suture. The muscular functions return much more rapidly after incomplete nerve lesions.

Sensory symptoms.—When a sensory nerve or the sensory fibers of a mixed nerve are cut across we might expect to find absence of all sensation in the cutaneous and deep structure which are supplied by that nerve; but by the ordinary methods of testing this is rarely found, as the sensory loss which is revealed is generally much less extensive than the anatomical distribution of the nerve. This has been generally explained by assuming that the nerves overlap or anastomose, or that the neighboring end organs take up sensibility from the anesthetic region. But the explanation has been afforded by the brilliant work of Head and his colleagues. These have shown that the afferent fibers may be divided into three systems. The first system subserves deep sensibility, which is conveyed, as Sherrington has shown, by the afferent fibers that run from the muscles, tendons, and joints, and which escape when only cutaneous sensory nerves are injured. Even when the muscular branches are involved this form of sensibility may not be quite abolished, as its fibers have wide anastomoses and often join the tendons and muscles high up in the limbs. Its function is the appreciation of pressure, of stimuli that produce deformation of structure, and of any change in the position and condition of the joints and muscles. It is the system which subserves the sense of position. It is owing to the persistence of deep sensibility that the statement is often made that no sensory loss ensues on the section of a cutaneous nerve, as the area of its distribution remains sensitive to even light pressure by a finger or pencil point.

The second system, to which the name protopathia is given, conveys painful cutaneous stimuli and the appreciation of the major degrees of temperature. But in the absence of the third system the

pain produced by a pin prick or other means is not localized, but radiates widely over the affected area and causes an unnatural amount of discomfort and an almost uncontrollable desire to withdraw the part from the stimulus. Although it is through this system that major degrees of heat and cold are appreciated, minor degrees of temperature can not be recognized when it is alone present, and the appreciation of degrees of heat and cold is lost. The third system, which has been called epicritic, responds to light touches and to the minor degrees of temperature, which produce the sensations called "warm" and "cool." It is only when epicritic sensibility is present that the point of skin touched can be accurately localized and that two points at a normal distance apart can be discriminated when applied simultaneously.

In the sensory disturbances that result from section of a peripheral nerve the different varieties of sensibility are lost according to their arrangement in these three systems; that is, sensibility to light touch and the minor degrees of temperature disappear together, and the appreciation of the major degrees of temperature with insensibility to painful cutaneous stimuli.

When the condition of sensation on the hand is examined after section of the ulnar nerve it will be seen that there is complete loss of all forms of sensation only on the little finger and on a variable, but small, area on the ulnar border of the hand; that is, it is only here that both the protopathic and epicritic sensibilities are absent, but on the rest of the cutaneous distribution of the ulnar nerve light touch and the intermediate degrees of temperature, the epicritic sensibilities can not be appreciated, and although painful stimuli may be recognized they can not be accurately localized, but radiate widely and give rise to unnatural discomfort. It is evident that though there may be a considerable overlap between the fibers of the ulnar and median nerves which conduct protopathic sensibility, the loss of epicritic is practically limited by the anatomical boundary of the nerve distribution. With other nerves there may be an overlapping of epicritic sensation too. When the fibers are injured in a plexus the area of loss of protopathic sensibility may almost equal in extent that of the epicritic, while if due to lesion of the dorsal spinal roots, the area insensitive to a pin prick may actually exceed that insensitive to light touch. The more closely a peripheral nerve represents the supply of one or more posterior roots the more nearly will the loss of protopathic coincide in distribution with the loss of epicritic sensibility. Head's conclusions have been contested by Trotter and Davies and others.

When a nerve is not completely divided, or if only its functional continuity is affected, as by bruising or compression, the condition of sensation is very variable. Sometimes every form of sensibility is

lost for a time, but often pain perception remains intact and loss of epicritic sensibility may be the only sign of the injury, or after slight injuries there may be no absolute loss of sensation, though the patient may be conscious of an altered sensibility in the area of the nerve.

The recovery of sensibility in the area of a divided nerve which has been sutured and is regenerating takes place in a definite and constant manner. The first change, generally observed after about two to three months, is a gradual diminution of the area of total analgesia commencing in the proximal parts of the area, and recovery of appreciation of major degrees of temperature, while the area of insensibility to light touch remains unaltered. At this stage, generally completed in about six months, there is still absolute loss of epicritic sensibility, although all the forms of protopathic sensation have recovered. The diffuseness and radiation of pain produced by a pin prick, or roughly handling, or a blow is so unpleasant at this stage that the complete absence of sensation may appear preferable to the patient. The return of sensibility to light touch and the minor degrees of temperature, as well as the power of discriminating between one and two points and of accurately localizing stimuli, rarely commences earlier than six months, no matter how favorable the nerve union may be, and is seldom complete within a year; but even for years careful examination may detect abnormalities.

After incomplete nerve lesions, on the other hand, the appreciation of pain and of light touch return at approximately the same time; it may be taken as an absolute rule that if the simultaneous recovery of protopathic and epicritic sensibility is observed within a few months of the injury the nerve has not been completely divided. Recovery of function is also much more rapid than after complete division of the nerve. It commences at a date which varies with the distance of the injury from the periphery, from about three weeks at the wrist to six months in the plexus and also with its degree.

Symptoms of irritation or perversion of function of sensory fibers are often a prominent feature of disease or injury of the peripheral nerves, especially when the lesion is only partial or when the central stump of a divided nerve is involved in scar tissue. Pain is the most important of these subjective symptoms. Irritation of a nerve by pressure or disease may give rise to severe pain; this is undoubtedly due to the excitation of the sensory fibers by the lesion; the abnormal excitation thus produced is conveyed by the ordinary paths to the higher sensory centers of the cerebral cortex, where it reaches consciousness; its origin is, however, misinterpreted and is referred to the sensory end organs of the irritated fibers, even although the area in which they lie be completely anesthetic—*anesthesia dolorosa*. Pain of this origin is often paroxysmal and is generally sharp, burning, or

darting; at times, however, it is of a dull aching or boring character; it may be referred to the skin or the deeper structures of the limbs.

Hyperalgesia is met with in regions in which epicritic sensation is lost, whole protopathic persists, but it often follows partial lesions where there is little or no disturbance of sensation. It not infrequently extends over the boundary of the anatomical distribution of the injured nerve and is often associated with severe spontaneous pain.

Weir Mitchell gave the name *causalgia* to a condition of severe spontaneous pain associated with hyperalgesia and tenderness and often with trophic disturbances. It is most frequently seen after bullet wounds; it never results from complete interruption of the continuity of the nerve and always disappears immediately the nerve is cut across for secondary suture. In these cases it is probable that normal stimuli of peripheral origin are augmented in their passage through the injured region, or it may be that the sensory end organs become hyperexcitable owing to a disturbance of their connection with their trophic centers. Hyperalgesia appears rarely if ever immediately after the nerve injury, generally not for a period of a few weeks.

Pain is uncommon after nerve injuries by modern high-velocity bullets; Olconomakes saw it very rarely accompanying peripheral palsies in the Balkan wars.

Various paresthesias, or a feeling of loss of feeling, or of weight or pressure, or, it may be, a sense of warmth or coldness, are frequently due to affection of the peripheral nerves which supply the region to which they are referred; they are generally, but not always, associated with some loss of sensibility. They are, on the whole, met with more frequently in the toxic degenerations of nerves than after traumatic or local lesions; they are probably due to a slight but persistent irritation of the sensory fibers. The most frequent form is generally described as a numbness or deadness of the part, but from the discomfort it causes it is evident that it is more than the consciousness of loss of feeling; it is rather an abnormal positive symptom, which may, even when there is no objective sensory disturbance, seriously interfere with the functions of the part. Even when paresthesias are due to the local injury of a nerve, they are rarely referred to the whole area of its anatomical distribution; in the limbs they tend to be more intense peripheralward.

Occasionally the irritation of one of the terminal branches of a nerve gives rise to pain extending over its whole protopathic distribution, or that of its roots, which may be accompanied by hyperalgesia, or by spasm or contractures of the muscles supplied by it. Neurotomy of the irritated branch gives instant relief.

Trophic and vasomotor symptoms.—The nutrition and conservation of all tissues of the body are to a certain extent dependent on the integrity of their connection with the central or autonomic nervous

systems, and when this is interrupted the isolated tissues may undergo certain changes. The most prominent of these is the atrophy of the muscles which follows the degeneration of the motor nerve fibers which innervate them, but the skin, subcutaneous tissues, and even bone may also undergo structural alterations.

It was for long discussed whether the influence of the nervous system on the tissues is exerted through special trophic fibers, or through the ordinary motor, sensory, or vasomotor nerves. The muscles are certainly dependent only on the integrity of their motor fibers, and the existence of special trophic fibers to other tissues has not been proved. The vasomotor nerves must, however, have a considerable influence on nutrition and growth; parietic vasodilatation follows section or injury of a nerve, and although there may be hyperemia and a slight elevation of the local temperature for a time, the part soon becomes cold, congested, and even cyanosed, owing to the slow circulation through the dilated vessels; and its inactivity leads to lymph stasis.

It seems probable that the trophic centers for the skin and other tissues lie in the spinal ganglia, and that the centrifugal conduction of trophic influence is a function of the sensory fibers; or more probably that the normal nutritional equilibrium is in some way determined by the sensory nerves. The strongest evidence of the trophic function of the spinal ganglia is that the acute changes in the skin which characterize herpes zoster are directly due to disease of them. The innervation of the blood vessels and of the sweat and sebaceous glands takes place through the sympathetic system; in the limbs, at least, these sympathetic fibers are intimately associated with those of motion and sensation.

Trophic changes in the skin vary with the rate of evolution and the degree of the nerve lesion; the more acute it is the more likely are trophic changes to be prominent. They may be met with either in areas of total anesthesia or associated with only partial disturbance of sensation. In areas of total analgesia the skin becomes thin and atrophic, and especially in the hands and feet, inelastic and tightly stretched over the part; owing to the cessation of sweat and sebaceous secretion it is usually very dry and may be either glossy and shiny or scaly when desquamation of the superficial layers of the epidermis is delayed. In this condition it is very liable to injury, and ulcers often develop, which, as a rule, first appear as blisters or bullæ; these often appear to be the result of the trophic disturbance alone, as they may occur apart from any assignable local cause or follow a slight bruise. Owing to the loss of sensation the skin is liable to suffer from neglect or unobserved injuries, and to this many of the trophic changes has been attributed. Head has observed that these

affections are generally coextensive with the area of complete analgesia and disappear with the return of protopathic sensibility.

Another form of cutaneous trophic disturbance which follows incomplete lesions of nerves and is always associated with spontaneous pain and hyperalgesia (causalgia) was first accurately described by Paget and Weir Mitchell, and by the former termed "glossy skin." It is seen chiefly on the fingers and hands; the skin becomes "smooth, hairless, almost devoid of wrinkles, glossy pink or ruddy, or blotched as if with permanent chilblains" (Paget). The subcuticular tissues shrink, and the skin, which appears tightly drawn over them, is often cracked and the epithelium is partially lost, so that the cutis is exposed in places. According to Weir Mitchell this condition is often attended with vesicles.

Changes in the growth and condition of the nails often follow nerve injuries. Retardation of growth has been frequently described, but Head has shown that this is independent of sensory disturbance and that want of movement owing to paralysis or the fixation of the limb is the main factor. Weir Mitchell observed remarkable alteration in the nails associated with glossy skin, which consisted of curving on their long axes, extreme lateral arching, and sometimes a thickening of the cutis beneath their extremities.

When several nerves of a limb are damaged the bones are liable to become fragile, and if it occurs in early life their growth may be retarded. Acute and extensive nerve lesions may lead to swelling of and effusions into joints in the paralyzed region. Another form of chronic joint change occurs chiefly in the fingers and wrists; it commences with pain and periarticular thickening and may ultimately produce ankylosis.

The treatment of local nerve lesions.—This naturally depends on the nature and the degree of the lesion, but there are certain general lines which should be invariably followed. *In every case the chief aims should be to maintain the nutrition of the parts, to keep the paralyzed muscles relaxed, and to prevent the occurrence of contracture.*

If the nerve has been completely divided, its two ends should be sutured together at once. The prognosis when primary suture is possible is good, but it depends to a certain extent on the nerve injured, the nature of the lesion, the condition of the wound, and the distance of the injury from the periphery; the muscular functions generally return within nine months, but sensation is rarely perfect for two or three years. Often, however, the nerve injury is not observed, or the patient does not come under treatment until after a considerable time; then secondary suture must be performed. The outlook in these cases is not so favorable as after immediate suture, but the time after the injury at which the operation is undertaken,

certainly up to three years, seems to have little influence on the recovery (Sherren).

Much depends on the condition of the muscles; when these have entirely lost their galvanic irritability, complete recovery is probably impossible; and according to Warrington and Jones, when the paralysis has lasted several weeks, return of full power can not be expected unless paralyzed muscles have been kept relaxed. It is important that not merely should all scar tissue around the ends of the nerves be removed, but that these should be freshened up and brought into accurate apposition. It is occasionally impossible to bring the divided ends together; then Assaky recommended imposing catgut threads between them to form a scaffold along which the regenerating axis cylinders may grow, and Vanlair has introduced the method of interposing a tube of decalcified bone or a piece of a small artery or vein for the same purpose. Nerve transplantation, however, seems to give more favorable results in such cases, but, as Merzbacher has shown, the portion of nerve inserted can take an active part in regeneration only when it is taken from the same animal or an animal of the same species; if taken from another species it immediately necroses and can consequently at the most act only as a scaffold. In other cases it is preferable to anastomose the peripheral end of the paralyzed nerve into a neighboring healthy trunk, or end to end with a flap raised from the sound nerve. In every case the exact condition of the injured nerve should be examined under an anesthetic if there is complete reaction of degeneration in the muscles it supplies after 14 days.

Tendon transplantation is occasionally necessary, especially when there is such complete degeneration of the paralyzed muscles that recovery after secondary suture of the nerve can not be expected. The results obtained are often favorable. It is interesting that after nerve anastomosis and tendon transplantation nerve centers in the cord can acquire new functions or adapt themselves to new conditions.

Whatever surgical treatment is adopted, it is important to keep the paralyzed muscles relaxed and prevent shortening and contracture of their antagonists. If the extensors of the wrist and fingers are paralyzed, their joints must be kept fully extended by a splint along the forearm and hand; if it is the flexors of the forearm, the elbow should be held flexed in a sling. But the splint or other apparatus used should be removed frequently and massage and passive movements carried out.

The next aim should be to maintain the paralyzed muscles in as good a state of nutrition as is possible, and for this electrical treatment is usually employed. If the paralysis is not complete, and if some excitability to the interrupted current remains, faradism may

be employed, but it is of little use if the muscles will not react to it, although, according to Mann, regular treatment raises the excitability of both nerve and muscle and increases the flow of blood and lymph. Galvanism is more effective when the muscles will not react to faradism, but the current employed should not be too strong.

Systematic and energetic massage is probably more effective than any form of electrical treatment and should be combined with it, but the region of the nerve injury should not be forcibly handled. Passive movements are always advisable, and the patient should make efforts to use the paralyzed muscles as soon as any return of power sets in.

SPINAL NERVES.

The cervical plexus.—Owing to its deep position among the muscles of the neck the cervical plexus is rarely injured or affected by disease. The branch which is of most clinical importance is the phrenic nerve. Its paralysis may be due to a lesion in the ventral horns of the cord at the level of its origin, to an intraspinal hemorrhage or tumor, or to syringomyelia. Duchenne first described it in progressive muscular atrophy. It is most frequently caused by involvement of the third and fourth cervical roots in meningeal or cerebral disease, especially in spinal caries and syphilitic pachymeningitis. Owing to its deep position the nerve trunk is relatively immune from trauma, but it is occasionally injured by wounds or operations. It has been repeatedly observed after local anesthesia of the brachial plexus. Within the thorax it may be compressed by tumors, aneurisms, or enlarged glands. Unilateral paralysis can be often attributed only to a local neuritis, perhaps following exposure to cold. Bilateral palsy sometimes occurs in multiple neuritis, especially in that form which follows the acute infective disease, in lead poisoning, and in tabes dorsalis (Gerhardt).

The diaphragm is one of the most important of the respiratory muscles; when the patient is at rest its inactivity may give no trouble, but dyspnea is easily produced by exertion or when respiration is interfered with by disease of the lungs or pleura. If bilateral paralysis sets in suddenly there may be considerable dyspnea and even cyanosis for a time, but it is quickly relieved by the activity of the accessory muscles. Several cases have been recorded in which accidental injury of one phrenic nerve during operation in the neck has led to a fatal result, but Schroeder and Green, who analyzed these cases, came to the conclusion that death was generally due to some other cause.

When there is complete bilateral paralysis the upper part of the abdomen is no longer protruded with each inspiration, but sinks in as the diaphragm is drawn upward by the negative pressure in the

thorax; on palpation the descent of the liver and spleen can be no longer felt, and the patient is unable to expand the abdomen by taking a deep breath. The movements of the thorax are consequently often increased, and the excessive movement of the lower part of the thoracic cage may draw the abdominal wall tense; this must not be mistaken for the effect of the descent of the diaphragm. If the paralysis is not complete, the protrusion of the abdomen in inspiration can be easily resisted by pressure. Owing to inability to take a deep breath there is difficulty in coughing, and the patient can not spit out with the normal force nor sneeze properly, and there may be difficulty in defecation. Pulmonary symptoms are the most important complication; owing to the relative immobility of the bases of the lungs they may have become dangerously congested.

The symptoms of unilateral paralysis are generally slight and frequently escape observation. but the deficient movement of one side can be definitely determined by a radiograph or by the direct observation of the movements. The electrical excitability of the phrenic nerves may be examined; they are easily stimulated in the neck between the sternomastoid and scalenus anticus muscles and above the omohyoid. The phrenic nerves also convey sensory fibers to the pleura, pericardium, and diaphragm, but sensory symptoms have been rarely referred to their disease: in a few cases, however, patients have complained of pain in the mediastinum and in the region of the diaphragm.

The diagnosis of diaphragmatic paralysis is not always easy; the examination of the electrical excitability of the phrenic nerves may be very important when other signs are not conclusive. In the majority of the cases the condition is only part of a general peripheral neuritis, or of poliomyelitis. Otherwise, bilateral paralysis is generally due to some disease in the spinal cord or meninges; it can then be rarely an isolated symptom. The vertebral column should be carefully examined and the existence of tumors in the neck and mediastinum excluded. Paralysis must be distinguished from immobility of the diaphragm owing to diaphragmatic pleurisy or peritonitis, or to large pleural effusions. Or the diaphragm may be weak owing to secondary degenerative changes. Acute fatty degeneration of its muscle fibers frequently occurs in diphtheria, and may enfeeble, but rarely paralyzes the action of the muscle.

Treatment.—This should be directed to the removal of the cause if it is possible, but it is rarely so unless it is due to an operable tumor either in the vertebral canal or neck. Otherwise the chief aim should be to avoid all pulmonary complications and save the patient from exertion. If there is reason to suspect a local neuritis. warm

fomentations and counterirritation may be applied over the lower part of the anterior triangle of the neck. Electricity, especially the faradic current, has been employed with apparently some effect; the one electrode should be placed over the phrenic nerve immediately behind the sternomastoid muscles in the neck, the other over the epigastrium.

Long thoracic nerve.—Isolated paralysis of this nerve is rare. Steinhäusen was able to collect records of only 29 pure cases. It is more frequently found in association with palsy of other muscles of the shoulder girdle, and it is often seen in progressive muscular atrophy and in the muscular dystrophies. The nerve may be injured in the neck by blows or perforating wounds, or by direct pressure from a heavy weight carried on the shoulder, and it occasionally happens that it is bruised by the forcible contraction of the scalenus medius muscle through which it passes, or by excessive stretching of the nerve when the arm is raised above the head, as in painting a ceiling, climbing hand over hand, or hanging suspended by the arms. In the axilla it may be injured by a perforating wound or by operation. Paralysis of the serratus magnus has also been observed after infective disease, and with acute arthritis of the shoulder joint. In some cases it has been apparently due to a local neuritis following exposure to cold. It is much more frequent in strong, muscular men than in women and is more common on the right side. Its paralysis produces very little deformity while the arm is at rest; the scapula may stand slightly higher than normal, with its inferior angle slightly approximated to the vertebral column and separated from the chest wall. When the arm is moved forward into the horizontal position or pressed forward against resistance, the scapula, no longer held against the thorax by the serratus, is rotated on its vertical axis, so that its vertebral border projects backward and appears winged. This deformity is almost pathognomonic of serratus palsy. There is also difficulty in raising the arm above the horizontal, as while the arm is normally abducted from the side as far as the horizontal level by the deltoid alone, its further elevation is brought about by rotation of the scapula chiefly by the serratus magnus. The latter part of the movement is consequently lost when this muscle is paralyzed, but not invariably, as it can be sometimes carried out by contraction of the middle fibers of the trapezius. There is often diminished muscular power in the whole arm, which disappears when the scapula is firmly bound to the trunk. Slight scoliosis is frequently observed in cases of serratus palsy; it is probably due to an attempt to reestablish the equilibrium upset by the malposition of the shoulder. When the paralysis is due to a neuritis its onset is often accompanied by severe neuralgic pains in the supraclavicular region, which may radiate up the neck, behind the scapula and even

into the arm, but no loss of sensation results from an isolated paralysis of the long thoracic nerve.

Treatment.—This must be conducted on the usual lines; it is important to prohibit work or exercise which must produce pressure or strain on the nerve. When the palsy is incurable, the humeral attachment of a portion of the pectoralis major may be inserted into the serratus magnus.

The suprascapular nerve.—Isolated paralysis of it is very rare; Fischler was able to collect only 14 cases. In half of these it was due to direct or indirect trauma, in others apparently to a local neuritis; it may be caused by the pressure of a heavy weight carried on the shoulder or by a fall of the shoulder or the outstretched arm. Its most prominent symptom is flattening of the infraspinous fossa and weakness of outward rotation of the humerus owing to the atrophy and palsy of the infraspinatus; this movement is not, however, completely absent, as the teres minor and the posterior fibers of the deltoid can execute it, but only very feebly. According to Duchenne, the chief function of the supraspinatus is to act as an elastic ligament in keeping the head of the humerus in close apposition to the glenoid cavity; when it is paralyzed, the humerus falls away and the movements of the shoulder joint are impeded, especially abduction and elevation in the sagittal plane. In every case of its paralysis the patient has complained of weakness and fatigue in the shoulder and of the inability to carry weights. No definite disturbance of sensation has been detected with suprascapular palsy, but its onset may be accompanied by pain in the shoulder girdle.

The circumflex nerve.—Paralysis is most often due to such injury as a blow or fall on the shoulder, dislocation of this joint, or fracture of the upper end of the humerus. Occasionally it is caused by the pressure of a crutch or by lying for long on the shoulder in deep sleep or in an unconscious state; in those cases in which paralysis follows prolonged operations under anesthesia the lesion is evidently due either to compression or undue stretching of the nerve. Similarly paralysis of the circumflex has been frequently observed in miners who work lying constantly on the left side. Local neuritis may be due to exposure or to extension of inflammation from arthritis or from disease in the axilla; neuritis has also been observed in infectious diseases and in diabetes. It is noteworthy that Bernhardt and Buzzard have observed isolated paralysis of the circumflex nerve in lead poisoning.

A complete lesion of the nerve leads to complete paralysis of the deltoid, except of a small number of its anterior fibers, which are supplied by the anterior thoracic nerves. Abduction and elevation of the arm in any plane are consequently impossible, except by rotation of the scapula by the trapezius and serratus magnus. The

supraspinatus may also aid in this movement. The shoulder joint becomes relaxed, and owing to the wasting of the deltoid its shape is altered. The paralysis of the *teres minor*, which depresses the arm and rotates its outward, is less prominent. The onset is generally accompanied by much pain. Objective sensory disturbances are not constant, but in typical cases the area of anesthesia to light touch occupies an oval area on the outer side of the arm extending from the level of the acromion process rather more than half way to the elbow; the loss of sensibility to pain and the extremes of temperature is less extensive. When the paralysis lasts for long there is danger of adhesions forming in the shoulder point, and ankylosis may occur. In obstinate cases good results have been obtained by transplanting the clavicular portion of the trapezius and pectoralis major into the deltoid and by grafting the subscapular nerve into the circumflex.

A little care and examination of the electrical reaction of the muscle serves to distinguish primary joint disease with the secondary wasting of the muscle from circumflex paralysis.

The musculocutaneous nerve.—It is rarely paralyzed alone. Bernhardt collected only 14 cases, but as it is not infrequently associated with lesions of other nerves it is important to recognize its symptoms. It is generally due to a blow on or compression of the arm or to fracture or dislocation of the humerus. When the lesion is complete the biceps and coracobrachialis are absolutely paralyzed, as well as the greater portion of the brachialis anticus. Flexion of the elbow is consequently impossible when the forearm is supinated, but it can be carried out feebly and in limited range by contraction of the supinator longus when the forearm is pronated. Sensory disturbance is limited to the radial side of the forearm and the thenar eminence.

The musculospiral or radial nerve.—This is probably paralyzed more frequently than any other nerve in the arm. It may be injured in the axilla by dislocation or fracture of the upper end of the humerus, or involved in callus formation or by the pressure of a crutch. It is occasionally compressed by the head of the humerus when the arm is kept full abducted and extended during operations under anesthesia (Braun). But it is much more frequently injured during its course round the humerus, very often by the pressure to which it is exposed when a person sleeps on a hard or uneven surface with the arm beneath his body or with the weight of the head resting on the outer surface of the arm. This occurs so often in a drunken sleep that it has been assumed that chronic alcoholism predisposes to it by lowering the vitality of the nerve (Oppenheim). Gowers has pointed out that it may be injured as it passes through the triceps by a sudden violent contraction of this muscle. Its paralysis

has followed the use of an Esmarch's bandage on the arm, and it sometimes results from the injection of ether. A local neuritis due to cold is occasionally assumed to be the cause. When palsy develops during an acute illness it is more probably due to pressure as the patient lies in a semiconscious or delirious state. The affection of some of its fibers is a characteristic feature of lead palsy.

Symptoms.—These depend on the site and the severity of the injury. When the lesion is in the axilla all the muscles supplied by it are paralyzed and the patient is no longer able to extend the elbow and wrist or the fingers or thumb at their basal joints, or to supinate the forearm, except by the biceps. If the nerve is injured, as it is more frequently, on the outer side of the arm, the extensors of the elbow and more rarely the supinator longus escape, but wrist drop, which is the characteristic feature of the palsy, is present. The thumb can not be abducted or extended, but its other movements are intact. Although the flexor muscles are not affected, the hand grasp is considerably weakened owing to the mechanical disadvantage at which they work when the wrist is not held straight by the normal action of their antagonists: with the hand passively extended, its grasp is normal. As the arm hangs by the side the forearm is generally pronated, and becomes more fully so when the hand grasps any object, owing to the unresisted pronation action of the flexors. The power of supination with the elbow extended is completely lost, but when it is flexed the biceps can supinate the forearm. If the nerve is injured in the forearm, the supinators and even the extensors of the wrist may escape. Paralysis of the supinator longus produces slight weakness of flexion of the elbow.

Sensory symptoms are very variable; with the onset there may be subjective sensations of numbness and tingling in its cutaneous distribution, generally most pronounced on the radial border of the hand. In incomplete lesions, the sensory fibers are, according to the general rule, much less affected than the motor, and even with complete paralysis of the muscle there may be no loss of sensibility; when present the anesthesia is generally most marked over the radial branch. Trophic changes are rarely prominent. In the pressure palsies there may be little or no atrophy of the paralyzed muscles. Occasionally a prominence develops on the dorsum of the hand, which is due either to the swelling of the sheaths of the extensor tendons (Gubler) or to overflexion of the carpus. There may be slight effusion into the carpal joints, and adhesions may form in them. The electrical reactions of the nerve and the muscles it supplies are extremely important; in the pressure palsies the nerve may be inexcitable from above the lesion, while in the portion below it and in the muscles it supplies normal responses can be obtained; the

conduction of volitional and electrical impulses is thus interrupted by a lesion which is not of sufficient intensity to damage the continuity or vitality of the nerve fibers. If the lesion is more severe there must be partial or complete reaction of degeneration in the muscles it supplies.

Paralysis of this nerve is generally easily recognized, but, as Gowers points out, the fact that it produces loss of extension of the limb at all its joints may lead to error, as palsy limited to a single function suggests central disease; the absence of sensory loss and of change in the electrical excitability of the muscles may increase the risk of error. In lead palsy it is the musculo-spiral groups of muscles which are chiefly involved, but the affection is almost invariably bilateral, the onset is usually slow and unconnected with trauma, the supinator longus, as a rule, escapes, and the reaction of degeneration appears early in the paralyzed muscles.

Treatment.—This must be conducted on the usual lines. In the pressure palsies the application of the galvanic current for 20 to 30 minutes at a time is of value; the cathodal electrode should be placed over the seat of injury, the anode distal to it, and the strength of current slowly increased until the patient feels it distinctly.

The median nerve.—Owing to its deep position among the soft tissues of the arm this nerve is much less liable to injury than the musculo-spiral. In the axilla and arm it is usually injured by fractures and dislocations of the humerus and occasionally by the pressure of a crutch, but the most frequent cause of its paralysis is a wound on the palmar surface of the wrist. Occupation palsies frequently involve some or all of the hand muscles supplied by the median nerve; it has been repeatedly observed in laundresses, joiners, milkmaids, and cigarette makers. The exact nature of the lesion is doubtful; often it is undoubtedly due to a neuritis set up by pressure, but in other cases the muscular palsy and atrophy seem to be the direct result of the stress of overwork. Drummer's palsy affects chiefly the thumb muscles, but seems to be generally due to rupture of the tendon of the extensor longus pollicis (Heinicke).

When the nerve is damaged above the elbow the power of pronating the forearm is lost, flexion of the wrist is feeble and incomplete, and, as it can be performed only by the ulnar flexor, the hand is strongly deviated to the ulnar side. Flexion of the interphalangeal joints is also lost except that of the distal phalanges of the two ulnar fingers, which can be still bent by the unparalyzed part of the flexor profundus. The flexion of the fingers on the metacarpus is unaffected, as it is performed by the interossei. The unopposed extensor action of the latter muscles at the interphalangeal joints may lead to their hyperextension. The thumb is kept extended and adducted by the muscles which remain, and it can not be opposed nor its distal

phalanx flexed; its metacarpal bone comes to lie in the same plane as that of the fingers, like the thumb in the ape. Owing to paralysis of these movements of the fingers and thumb there is considerable difficulty in firmly grasping any object and in employing the hand in any work. When the lesion affects the nerve in the forearm, after its branches to the pronators and flexors have been given off, pronation and flexion of the wrist and fingers may be intact; then it is chiefly the thumb movements which are lost. Bernhardt and Head have pointed out that the branch which supplies the muscles of the hand may leave the main stem of the nerve in the lower part of the forearm and thus escape injury when the wound is at the wrist. When the lesion of the nerve is severe, prominent atrophy of the muscles of the thenar eminence results, and of the flexor surface of the forearm if it is above the elbow. There may be considerable paresthesia in the cutaneous area of the nerve in earlier stages of the paralysis; the occupation palsies and neuritis are generally accompanied by troublesome pain. A complete lesion of the nerve may lead to troublesome trophic disturbance of the skin and nails in the area where protopathic sensibility is lost, and to vasomotor paresis and cessation of sweating in the radial part of the palm.

The ulnar nerve.—This may be injured alone or with other nerves in the axilla or upper arm by dislocation or fractures of the humerus, or involved in callus formation; occasionally it suffers in crutch palsy. It is much more frequently damaged at the elbow joint by dislocations or fractures; occasionally ulnar paralysis develops slowly at long periods after elbow injuries, owing probably to the pressure of fibrous adhesions or excess of callus on the nerve, and in a few cases it has been due to traumatic or synovial cysts in this region. More rarely the curious condition of dislocation of the ulnar nerve from its groove on the posterior surface of the internal condyle is met with; it probably occurs only when the internal condyle is badly developed. Paralysis due to direct pressure from without is rare; it is occasionally due to pressure on the elbow during sleep, especially in emaciated subjects or during infective illness. According to Braun, however, these sleep palsies are to be attributed to pressure of the head of the humerus on the nerve in the axilla when the arm is abducted and extended. But undoubtedly wounds in the wrist, which may either injure this nerve alone or the median nerve and flexor tendons as well, are the most common cause of ulnar palsy. Primary neuritis is rare; a few cases of syphilitic neuritis are described, and leprosy has a predilection for this nerve.

When the nerve is injured at or above the elbow the power of flexion of the hand is very feeble, and when attempted the hand is deviated radialward by the radial flexor; the wrist becomes hyperextended when the fingers are straightened owing to the palsy of

its ulnar flexor. The movements of the little finger are lost, the middle and ring fingers can not be flexed at their distal joints, and owing to the paralysis of the interossei the basal phalanges of all the fingers can not be flexed or the middle or distal phalanges fully extended. Adduction and abduction of the fingers are also impossible. When the injury is situated in the lower part of the forearm the fibers to the flexor profundus digitorum escape and the interossei and thumb muscles are alone paralyzed; then, owing to the unopposed contraction of the long extensors and flexors, the hand becomes clawlike (*main en griffe*), with the first phalanges hyperextended and the interphalangeal joints flexed. As the first two lumbricales escape, the deformity is generally not so pronounced as it is in progressive muscular atrophy (Gowers). Adduction of the thumb is also lost. The muscular atrophy which follows severe lesions of this nerve is very typical; the hypothenar eminence disappears, the palm becomes hollow, and the interosseal spaces sink in. When the nerve is severely damaged there is complete loss of sensation in the little finger and over a variable extent of the ulnar border of the hand; on the ring finger and the rest of the ulnar cutaneous distribution sensibility to light touch and the intermediate degrees of temperature are alone affected. As the dorsal cutaneous branch separates from the nerve in the middle third of the forearm it may escape in wounds at the wrist. Dupuytren's contracture of the palmar fascia has been observed after neuritis of the ulnar nerve, and De Leon has described contractures of the ulnar portion of the flexor profundus digitorum, owing to which the three ulnar fingers were bound down in the maximal flexion position.

The chief danger of error in diagnosis is of confusing central and peripheral lesions, as the ulnar nerve contains almost all the root fibers of the eighth cervical and first thoracic segments of the cord.

Paralysis of the brachial plexus.—The brachial plexus is formed by the anterior primary divisions of the lower four cervical and the first thoracic roots; it contains all the fibers which supply the muscles of the shoulder girdles and upper extremities, as well as the sensory fibers to almost the whole of the arm. By division and secondary anastomosis of these roots the three main cords of the plexus are formed. The outer cord receives the ventral trunks of the fifth, sixth, and seventh cervical roots; the inner contains the ventral trunk of the eighth cervical as well as the whole of the first thoracic root, and the posterior cord is made up of the dorsal trunk of the four lower cervical roots. This is the most common form of the brachial plexus, but it is liable to variation, for the fibers concerned in any single function do not constantly leave the cord by the same roots; but although fibers may alter their position relative to the vertebral column, they always maintain their position in relation to

other fibers (Herringham). The whole plexus may be shifted up—high or prefixed type—or downward—low or postfixed type—and the extreme variations between the high and low form of plexus may amount to nearly a whole root (Harris). Paralysis may be due to injury or disease of the nerve fibers in the roots, in the plexus, or in the nerve trunks and branches. Two types of plexus paralysis merit special description.

Duchenne-Erb palsy, or the upper-arm type.—In this form the deltoid, biceps, brachialis anticus, and supinator longus are generally paralyzed together, and frequently also the supinator brevis and supra and infraspinati; more rarely other muscles of the shoulder girdle and the radial extensor of the wrist and the pronator radii teres. The lesion to which it is due involves the fifth and sixth cervical roots either before or immediately after their union, or occasionally the fifth root alone; when caused, as it frequently is, by a blow or fall on the shoulder, it has been assumed that these roots were compressed between the clavicle and the transverse processes of the lower cervical vertebræ, or the first rib, but it seems more probable that the lesion is a rupture of some or all of the fibers of this portion of the plexus owing to excessive stretching and tension. In many cases at least the root fibers themselves are ruptured or are torn out from the spinal cord. This may be due to any cause which increases the distance between the shoulder and the head and neck. The vulnerability of the two upper roots of the plexus to this form of trauma is explained by the fact that they bend downward immediately on their exit from the spine, so that the extravertebral portion of each root forms an angle open downward with the portion which lies in the intervertebral foramen, while in the lower two roots this angle is open upward, and in the seventh root there is little or no bend.

In severe cases the arm hangs by the side and can not be abducted because of the paralysis of the deltoid or rotated outward at the shoulder if the infraspinatus is affected. Flexion of the elbow is impossible, owing to the paralysis of the biceps, brachialis anticus, and supinator longus, or it can be effected through a small range by the pronator radii teres and the flexors of the wrists. Supination of the forearm is always weakened by the loss of power in the biceps, and it may be impossible if the supinator brevis is also affected. Sensory symptoms are absent in the slighter cases, but there may be paresthesia and loss of sensation on the radial side of the arm and forearm. Even complete section of the anterior primary division of the fifth, and sometimes of the fifth and sixth, roots may produce no sensory loss (Sherren). Erb has shown that all the muscles paralyzed in this type of plexus palsy may be made to contract by electrical stimulation over a point in the neck 3 cm. lateral to the

sterno-mastoid and the same distance above the clavicle; the loss of excitability of the nerve trunks from this point is an important sign of the upper arm type of plexus palsy.

Klumpke palsy, or the lower-arm type.—This form of paralysis, in which the eighth cervical and first dorsal roots are involved, is generally met with only as the residue of a more extensive lesion, but it may be due to compression by a tumor in the neck or a growth in connection with the lung or vertebral column. Owing to the deeper position of these roots, they are rarely injured by trauma. The palsy is characterized by an atrophic paralysis of the intrinsic muscles of the hand and generally of some of the forearm muscles, especially the flexors, with a certain amount of loss of sensation on the ulnar fingers and the ulnar border of the hand and forearm. Frequently, too, there are pupillary symptoms, due to injury of the sympathetic fibers, which leave the cord in the first thoracic root; but as these branch off from the root immediately outside the intervertebral foramen, they are affected only when the inner portion of the root is damaged. When they are injured the palpebral fissure is narrowed, the pupil contracted, and there may be a slight degree of exophthalmos on the same side as the arm palsy. Vasomotor paresis on the same side of the face occurs probably only when the second and third thoracic roots are injured (Klumpke). When the small hand muscles are alone paralyzed the typical deformity of claw-like hand results; if the flexors of the fingers and wrist are also powerless, the wrist may be hyperextended, owing to contracture of the extensors.

These traumatic plexus palsies are frequently followed by severe spontaneous pain in the arm, probably owing to the constriction of the nerve fibers in scar tissue, and often by trophic disturbances.

Nerves of the lower limbs.—These are much less frequently involved in injuries or affected by disease than those of the upper extremities.

The anterior crural nerve.—Isolated palsy of this nerve is rare, but it may be due to compression by abdominal growths or by a psoas abscess, or to injury by fractures of the upper end of the femur or of the pelvis, or disease of these bones. Primary local neuritis is seldom seen, but occurs occasionally in diabetes. This nerve, either alone or with the obturator, may be injured during parturition; this probably occurs with greater frequency than is recognized, owing to the rapidity with which it recovers from slight compression and to the fact that its symptoms may not be noticed while the patient is confined to bed. Ernst has observed 30 cases in 800 births. The psoas muscle is paralyzed only when the nerve is damaged in the immediate neighborhood of the lumbar plexus; when the lesion is situated here the thigh can not be flexed on the abdomen, and if paralysis is bilateral the trunk can not be flexed on the thighs when

these are fixed; the patient is consequently unable to rise from the supine position. When the lesion is in the intra-abdominal portion of the nerve the iliacus alone is paralyzed, and flexion of the hip is only weak. The most prominent symptom is paralysis of the extensors of the knee, and the absence of the knee jerk in the affected limb. Paralysis of these muscles does not make standing or walking impossible, but contraction of its flexors must be avoided, as the patient can not resist flexion or actively straighten the joint. The paralysis of the pectineus and sartorius does not produce any other prominent symptom.

Its sensory branches arise in the upper part of the thigh; the middle and internal cutaneous are distributed to the lower two-thirds of the front and inner side of the thigh, the internal saphenous to the front and inner side of leg, and the inner side of the dorsum of the foot. Disturbance of sensation, paresthesia, or radiating pains may be present over these areas when the nerve is injured at or above the level of the groin.

The diagnosis is, as a rule, evident; the only risk is of confusing the marked atrophy of the quadriceps extensor group on the front of the thigh which occasionally results from inflammation of the knee joint with atrophy of these muscles, due to a nerve lesion; but in the former, although the electrical excitability of the muscles may be diminished, there is no reaction of degeneration. The ordinary lines of treatment must be followed; good results have been obtained by grafting the tendon of one of the flexors of the knee into the tendons of the quadriceps extensor when the paralysis is permanent.

The obturator nerve.—This is still more rarely injured alone. It may be injured during parturition, by intra-abdominal, or pelvic growths, or by an obturator hernia. When the muscles it supplies are completely paralyzed the limb can not be adducted, and although it can be raised by flexion of the hip, it can not be thrown across its fellow when the patient is seated on a chair. Owing to paralysis of the obturator externus, outward rotation is enfeebled, and inward rotation of the thigh is also weak, as the adductor magnus is paralyzed. Gait is not seriously interfered with. The disturbance of sensation is limited to a small area on the inner side of the lower half of the thigh.

Meralgia paresthetica is a condition characterized by paresthesia and pain, usually with slight objective disturbance of sensation, in the region supplied by the external cutaneous nerve on the front and outer side of the thigh. It occurs chiefly in middle-aged men, less frequently in women. Its etiology is obscure; in many cases there has been a history of trauma; probably the long course of the nerve through the fascia predisposes to its injury. In one case a localized.

perineuritis was found, but in other specimens there was no abnormality. In about one-sixth of the cases reported the condition was bilateral. In 1900 Schlesinger analyzed 122 recorded cases, and in the same year Musser and Sailer added 10 personal observations in a valuable contribution. The condition has been associated with flat foot. The symptoms vary greatly in intensity but the most common complaint is of abnormal sensations, of numbness, coldness, or tingling on the front and outer surface of the thigh. In other cases pain is the chief symptom; it may be very severe, but is usually felt only after walking or standing; it is probably due to constriction of the nerve as it passes through the deep fascia when the latter is tense. Occasionally it persists even when the patient is lying down. In the majority of the cases there is a point of tenderness just below the anterior superior iliac spine where the nerve pierces the fascia. Objective sensory disturbances are very variable, but often considerable; occasionally there is hyperesthesia. The symptoms are very intractable; rest may be necessary when there is much pain and massage, and the faradic brush over the course of the nerve may give relief. Resection of the nerve has cured some cases, but in others the pain has returned (Bramwell).

Similar symptoms have been occasionally observed in the distribution of the middle cutaneous branch of the anterior crural nerve, but generally associated with meralgia paresthetica. Lasarew, who has found the condition isolated, has given it the name meralgia paresthetica anterior.

Gluteal nerves.—Isolated paralysis of the superior gluteal nerve is uncommon; the muscles it supplies are the chief abductors and inward rotators of the thigh, and when they are paralyzed these movements are weak or lost. As the posterior fibers of the glutei rotate the limb outward this movement becomes weak. The interior gluteal nerve is rarely paralyzed alone; when this occurs the thigh can not be forcibly extended, nor the trunk straightened on the thigh when the lower limbs are fixed. Standing and walking on a level are not seriously interfered with, but the limb is of little use in ascending steps, and the patient has difficulty in rising from the sitting position.

The sciatic nerve supplies motor fibers to the hamstrings and to all the muscles below the knee, as well as the skin in the outer side of the leg and the whole of the foot, except a small part of the inner portion of its dorsum. Its main terminal branches are the external popliteal or anterior tibial nerve; these generally separate in the popliteal space, but are sometimes distinct from their origin in the plexus.

The external popliteal nerve may be injured by direct trauma in any part of its course, but it is especially liable to be bruised by a blow

or by pressure as it bends round the fibula. It has been occasionally torn or ruptured by violent extension of the limb, and paralysis has been frequently observed in laborers who work in a kneeling or crouching position; there it is probably due to compression of the nerve between the fibula and the tense tendon of the biceps cruris. A primary neuritis of this nerve is by no means rare, and even in a general neuritis its fibers seem especially liable to degeneration. Lead palsy is occasionally limited to its distribution, especially in children (Putnam), but in these cases the tibialis anticus escapes as a rule. Isolated paralysis of this nerve is sometimes seen in *tabes dorsalis*.

The whole limb must be unduly raised as it is brought forward in walking to enable the toes to clear the ground. Inversion and eversion of the foot are also weakened where there is a total paralysis. When the tibialis anticus is alone paralyzed the foot can be still flexed by the long extensors of the toes, but it is at the same time abducted; while if the function of the latter muscle alone is lost the active tibialis anticus inverts and adducts the foot. As Bernhardt points out, it not infrequently happens that the tibialis anticus escapes when the other muscles of this group are paralyzed. The paralysis of the peroneus longus is most evident when the ankle is extended, as the foot is then so strongly inverted by the unopposed action of its extensors that its outer border rests on the ground, and, as the inner part of the foot is not supported during extension, flat foot may develop. When all these muscles are paralyzed talipes equinus gradually develops, owing to contracture of their antagonists; if the paralysis of all the muscles is not equal in degree the foot may be at the same time either inverted or everted, according to the degree of the paralysis of the muscles with the opposing function. The toes may be permanently flexed by the contracture of the unopposed flexors and interossei. The sensory loss which results from a lesion of this nerve is limited to the outer side of the leg, the dorsal surface of the foot, and the dorsum of the first phalanges of the toes.

The posterior tibial or internal popliteal nerve.—Owing to its deeper course it is less liable to injury than the external popliteal, and its isolated paralysis is consequently rarer. A few cases have been recorded in which it has been injured by the tendons of the flexors of the knee when these muscles are forcibly contracted (Oppenheim), and it may be compressed by or involved in tumors, aneurisms, or inflammations.

The most prominent feature is inability to extend the foot or flex the toes, so that the patient can no longer stand on tiptoe or spring from the forepart of the foot in walking; if the paralysis is of long duration the unantagonized action of the flexors of the ankle produces talipes calcaneus, while the unopposed action of the peroneus

longus leads to eversion of the foot and increases the plantar arch. Flexion of the distal and middle phalanges of the toes is no longer possible, owing to paralysis of the long flexors, while the loss of the interossei, and of the adductors and abductors of the great and small toes makes lateral movements impossible. The unopposed contraction of the long extensors may lead to permanent overextension of the basal phalanges.

When the conduction of sensory impressions is completely interrupted there is loss of sensation on the outer side and back of the lower third of the leg, on the outer border of the foot, and on the sole and plantar surfaces of the toes, as well as in the dorsum of the distal phalanges. There may be trophic disturbances and ulcers may form.

Paralysis of the main trunk of the sciatic nerve may be produced by fractures of the pelvis or of the upper end of the femur, or by dislocations of the hip-joint; or the nerve may be compressed by tumors in the pelvis or invaded in the extension of septic processes from the surrounding tissues. Some or all of the fibers may be paralyzed during parturition, but the lesion is then generally situated in the lumbosacral plexus; the sciatic nerve of the child may be injured by traction on the leg in breech presentations. The symptoms of complete paralysis by a lesion near the sciatic notch are those of paralysis of its terminal branches, the internal and external popliteal nerves, with, in addition, palsy of the flexors of the knee. When the latter are powerless the limb must be held extended at the knee in walking, and it can be used only as a stilt; there is no power of movement at the ankle-joint. As all the sensory fibers enter its terminal branches, the loss of sensation in a complete sciatic palsy includes the outer side of the leg and the whole of the foot except a small area in the inner side of the dorsum.

The lumbar and sacral plexuses.—Isolated paralysis of the lumbar plexus is extremely rare and merits no further reference, but palsy of the whole or part of the sacral plexus is occasionally met with. It may be due to invasion or compression of some or all of its roots by tumors or inflammation, or the roots may be injured by pressure from the fetal head during birth. In the latter case it generally is only the fibers which enter the external popliteal nerve which suffer. This, it has been shown by Hunermann and Thomas, is due to the fact that the higher roots of the plexus, from which this branch receives the majority of its fibers, lie directly on the bone as they pass over the brim of the pelvis and are consequently more liable to suffer from compression than the sacral roots which are separated from the bone by the pyriformis muscle. For the same reason the superior gluteal nerve is often injured at the same time. But probably the most common causes of paralysis of these roots are malignant tumors

of the pelvis, or tuberculous caries, sarcomata or metastatic carcinomata of the sacrum, which either compress or invade these roots in the intervertebral canals. The symptoms are generally those of an incomplete sciatic paralysis, but if the upper roots are involved the outward rotators of the hip and the gluteal muscles are in addition paralyzed; or if the lower, there will be probably sensory loss in the distribution of the small sciatic nerve on the back of the thigh and on the buttocks and perineum.

Diagnosis.—The diagnosis of disease of the nerves of the lower limbs is as a rule easy, but different conditions with which a partial or complete paralysis of the sciatic nerve and its branches may be confused needs further consideration. The diagnosis is often greatly dependent on the history of the mode of onset of the paralysis and of its course; when it immediately follows an injury in the region of the nerve there can be little room for doubt if the symptoms correspond to the portion of the nerve injured.

Disease of the sciatic nerve and its branches must be distinguished from :

(1) *Lesions of the sacral plexus and of the extradural portions of the lumbosacral roots.*—When the disease is situated in the sacral plexus, muscles other than those supplied by the sciatic nerve are paralyzed, as the glutei, the obturator internus, the gemelli, and the quadratus femoris; and the anesthesia may extend to the back of the thigh and to the buttocks if the lower portion of the plexus is involved. A careful examination of the pelvis may reveal the presence of a tumor or of other disease. The extradural portions of the sacral roots are most frequently involved by tumors or disease of the sacrum; at first, as a rule only one root is affected and the earliest symptom is generally pain, which is often extremely severe, referred to the peripheral distribution of its sensory fibers, and paresis of the muscles supplied by it. The neighboring roots are subsequently paralyzed, and if the disease extends across the middle line motor and sensory symptoms may develop in the opposite limb. The distinguishing feature, as contrasted with a plexus or nerve paralysis, is that the motor and sensory symptoms correspond in extent with the distribution of the root fibers. Further, if the lower sacral roots are involved before they give off their visceral branches, the bladder and rectum are paralyzed; true sphincter paralysis never results from disease of the nerves.

(2) *From the lesions of the cauda equina* the paralysis of the nerves which spring from the sacral plexus may be distinguished by the fact that the symptoms in the former are always of the radicular and not of nerve distribution, that they are almost invariably bilateral, and that when the disease has advanced sufficiently far all the roots below the level of the intrathecal disease are generally involved. The sphincter functions, too, are almost invariably affected. It is

more difficult to distinguish between disease of the cauda equina and of the extradural portions of the spinal roots; in the latter condition, however, the symptoms are often uniradicular for a considerable time, as the sacral disease to which they are most commonly due will generally involve only one root at the first. Another point of distinction is that all the roots below the level of the affected one are not paralyzed in the latter condition, no matter how long the disease lasts, unless the sacral tumor extends into the vertebral canal and compresses the cauda equina, while when this is primarily affected by tumor or meningitis, all the roots which pass through the level of the disease are as a rule compressed.

(3) *From disease of the sacral segments of the cord* the diagnosis is easier. The symptoms, as a rule, develop more rapidly; they are almost invariably bilateral and are typically segmental in distribution, but all functions represented in the segments below the upper level of the disease are interfered with. If, however, the lower segments are not involved, the paralysis of the muscles which they supply is not associated with atrophy or change in the electrical reactions. The severe radiating pains, which are an almost invariable symptom of root lesions, are absent, and anesthesia develops earlier. The sphincter functions are generally seriously affected.

Sciatica.—This term is commonly applied to all affections of which the chief symptom is pain in the distribution of the sciatic nerve. Such pain may be of the nature of a neuralgia and unassociated with any disease of the nerve, or it may be due to a neuritis, or to compression of the nerve or its roots by tumors or by fibrous adhesions secondary to inflammation. It is unfortunate that the one term should be used for the symptoms of these different conditions, but it is, indeed, often difficult to differentiate between them. It is, however, important to separate the cases in which there is pain without any evidence of organic disease in the nerve from those in which sciatic pain is associated with symptoms of a nerve lesion, as anesthesia atrophic muscular paresis, change in the electrical reactions, and loss of the Achilles tendon jerk.

Etiology.—Males are affected much more frequently than females, in about the proportion of 5 to 1. It occurs more frequently in middle life, and very rarely, if ever, under 15 years of age. It has been attributed to almost innumerable causes, but exposure to wet and cold is generally the only apparent exciting factor; it may follow sleeping in a damp bed or sitting on a wet or cold seat. Gowers insists that many cases develop on a gouty diathesis; others undoubtedly follow spondylitis. The disease may be also due to trauma to the nerve, as by continuous pressure on the edge of a chair, a fall on the buttock, or injury in the neighborhood of the hip joint. Occa-

sionally an attack sets in after severe muscular exertion, but probably only in those predisposed to the disease. It occurs frequently in anemic and badly nourished subjects and in the course of chronic intoxications (alcohol), without any apparent exciting cause, and often after infectious diseases. Sciatica may be a symptom of diabetes, and is then usually bilateral. Quénu has shown that the pain may be due to the pressure of varicose veins on the nerve in the neighborhood of the sacrosciatic foramen; this generally occurs only in those who work all day standing erect.

Sciatic pain may be also due to the presence of tumors or inflammatory processes in the pelvis, or to a loaded rectum, which may either directly compress the nerve or affect the nutrition by venous stasis it produces. Finally, pain in the course of the sciatic nerve may be due to affection of its roots by disease of the sacrum or lesions of the cauda equina. Hysterical sciatica has been described.

The chief symptom is pain along the course of the nerve, or limited to one of its chief branches. The onset is occasionally sudden and associated with slight pyrexia and constitutional disturbances; but, as a rule, it sets in gradually with pain in the buttock or back of the thigh in movements or in postures which make the nerve tense or cause pressure upon it. In other cases the onset of the typical severe pain is preceded by slighter diffuse pain or a feeling of discomfort during walking or after exercise. The pain increases gradually in severity; it may be either gnawing and burning, or sharp and darting in character. As a rule, it is constant, but severer paroxysms occur, either spontaneously or excited by movement of the affected limb, and its intensity generally increases at night. It may be at first limited to one portion of the nerve, generally that in the upper portion of the thigh, but as the disease develops it usually extends along the whole length of the sciatic trunk and its branches. Often bouts of pain occur which shoot from the buttock down the limb; such attacks may be described by the patients in similar terms to the lancinating pains of *tabes dorsalis*. It is usually most intense in certain points, as over the sciatic notch, in the middle of the thigh, in the popliteal region, below the head of fibula, and behind the external malleolus; more rarely it is referred to the region of the posterosuperior iliac crest, or is most severe in the foot. The seat of the chief pain is often, however, variable in any case from day to day. It is generally more or less accurately limited to the course of the nerve, but in other cases it is referred to its whole cutaneous distribution. The most comfortable posture is lying on the back or on the affected side, with the thigh slightly flexed and the knee considerably bent, and when sitting the patient generally rests only on the tuber ischii of the unaffected side with the hip joint of the painful limb extended as much as possible. In walking the hip and knee are

held in moderate flexion with the foot extended at the ankle joint and only its toes and forepart touching the wound. Any sudden movement may bring on an attack of pain.

The disease is further characterized by the extreme tenderness of the nerve to pressure, rarely absent, except, according to Edinger, in those cases in which the sciatica is due to compression of the nerve by distended veins, and in which the spontaneous pain disappears when the patient lies at rest. In some cases the muscles of the limb are also tender to pressure. It is characteristic of the pain that it can be invariably produced by stretching the nerve; this can be most easily done by flexing the thigh with the knee extended or by extending the knee when the hip is flexed, *Lasegue's sign*, or, as Gowers has shown, by pressure on the nerve in the popliteal space as the patient sits in a chair with the knee flexed to a right angle; the pain which is produced by either of these means is felt not only at the point of pressure, but along the course of the nerve in the back of the thigh. Bechterew has pointed out that full flexion of the opposite hip when the limb is extended at the knee also often produces pain, but believes this indicates disease of the roots or of the cauda equina.

The pain is usually associated with paresthesia. The muscles supplied by the sciatic nerve and its branches often become flabby and undergo a slight degree of general wasting, even in cases which are not due to any organic lesion, when the disease is of long duration; but in this class of cases there is no change in the electrical excitability of the muscles. Severe reflex spasms of the limb and cramps in some of its muscles, especially in the calves, are not infrequently observed.

Slight trophic and vasomotor disturbances are occasionally met with, but usually only pallor, dryness, and coldness of the skin. In other cases there may be an increase of the surface temperature and of sweat secretion. Herpes has been observed. Scoliosis of the lumbar spine convex to the affected side is often seen, but its immediate cause is in dispute; it may be due to an attempt to spare the painful limb by tilting the center of gravity toward the opposite side, or to a relaxation of the lumbosacral muscles of the affected side. All explanations meet with the difficulty that the scoliosis is occasionally in the opposite direction, concave to the side affected. Kyphosis also occurs, but only rarely.

In addition to the pain and tenderness, evidence of organic disease is present in a considerable proportion of the cases. There may be diminution of cutaneous sensibility, but, as a rule, it is nothing more than a blunting of tactile sensation on the back of the leg and on the foot. When there is an organic nerve lesion the hamstrings and leg muscles and occasionally the glutei may be found not merely flabby but distinctly wasted and weak; but the feebleness of movement due to a true paresis must be distinguished from the reluc-

tance of the patient to exert full power owing to the fear of pain. The most certain indication is the presence of qualitative changes in the electrical reactions of the wasted muscles, which must be regarded as proof of the existence of an organic lesion of the nerve. The third sign of the existence of organic disease in the nerve is the absence of the Achilles tendon jerk. It seems very doubtful if this reflex ever disappears in the purely neuralgic cases; in fact, in many such cases it is very brisk or even exaggerated, as both the afferent and efferent paths of the reflex arc are contained in the sciatic nerve; its diminution or disappearance is one of the most delicate signs we possess of the presence of organic disease of the nerve; probably this may be excluded in all cases in which the reflex is undiminished. The reflex seems to be absent in about 30 to 40 per cent of the cases which are clinically regarded as sciatica (Strasburger).

In the great majority of the cases sciatica is unilateral, but it is occasionally bilateral in about 7 per cent of all cases, according to Gibson's statistics, though Hyde found it in 33 per cent of his cases. This, as a rule, indicates a general and not a local exciting cause; it occurs frequently in diabetes, and may be a part of an incomplete general neuritis.

Sciatica has been regarded by some recent authors as a disease of the dorsal roots of the cauda equina. Dubois pointed out that the hypoesthesia, when there is any, may correspond in extent to the distribution of one or more of the sacral roots, and Lortat, Jacob, and Sabareanu confirmed his observations. The sensory loss, according to these observers, most commonly coincides with the cutaneous areas of the last lumbar and the upper two sacral roots. In at least two of their six cases, however, there was an earlier syphilitic infection, and in one of these a lymphocytosis of the cerebrospinal fluid made probable the existence of a syphilitic meningitis. The fact that the small sciatic nerve and more rarely the anterior crural and the perineal nerves are occasionally affected simultaneously indicates that the disease is then not limited to the sciatic but probably affects the lumbo-sacral plexus or the spinal roots. In the latter case there is usually an excess of lymphocytes in the cerebrospinal fluid, and, according to Bonola, the fascia lata reflex, which is unaffected in sciatica, is lost.

Diagnosis.—The term sciatica is applied by custom to cases in which the symptoms are due to an organic affection of the nerve, as well as to those in which there is no evidence of structural disease. The first step must be, however, to separate these two classes, simple sciatica or sciatic neuralgia, in which there is spontaneous pain and tenderness of the nerve to pressure and to tension, but no pronounced sensory disturbance, degenerative atrophy of the muscles, or diminution of the Achilles tendon jerk; and organic sciatica or sciatic neu-

ritis, in which some or all of these signs of disease of the nerve are present. Some authors, as Gowers, regard all cases of sciatica with persistent tenderness of the nerve as neuritic, and consequently make simple sciatica or sciatic neuralgia very rare; but in facial neuralgia, in which there is certainly, as a rule, no disease in the nerve, its trunks may be quite as tender to pressure as the nerve in sciatica. Oppenheim and others have insisted that tenderness of the nerves is not a differential sign between neuritis and neuralgia. The pain of hip disease may radiate a short distance down the thigh; from sciatica it may be distinguished by the absence of tenderness in the nerve to pressure and tension, and the occurrence of pain on movement of the hip joint, and on pressure on the trochanter. Disease of the sacro-iliac synchondrosis may be more difficult to recognize.

Although many cases of sciatica are due to a neuritic or morbid process in the nerve, the presence of marked sensory disturbance and degenerative atrophy of the muscles, with the absence of the Achilles tendon jerk, should always raise the suspicion of more serious disease. If it is due to the compression of the nerve or its roots by a tumor in the pelvis, the sciatic trunk is not tender to pressure.

When the sacral roots are involved in disease of the sacrum or of the cauda equina, the symptoms are generally bilateral and more irregular in distribution, and the functions of the sphincters are, as a rule, affected; the pain is also generally referred to the cutaneous distribution of the affected fibers; the sensory or motor disturbances correspond to root distribution, and the nerve trunk is not tender.

In the early stages of tabes dorsalis the shooting pains may be limited to the sciatic distribution, but a careful examination of the case will generally reveal characteristic signs; in it the pain is almost invariably bilateral, and the nerves are not tender to pressure or stretching.

The pain of intermittent claudication occurs only after exercise and is generally most intense in the distal segments of the limbs and is not limited to the course of the nerves.

Prognosis.—In simple sciatica this is good as regards ultimate recovery, but it is extremely difficult to predict the duration, although as a general rule it is proportional to the severity. Cases in which pain is associated with signs of an organic nerve lesion are less favorable than the uncomplicated neuralgic cases. The outlook is less favorable in cases of long duration and where adequate treatment is not possible. Where the sciatic pain is due to some lesion extrinsic to the nerve which compresses or injures it, the prognosis is naturally dependent on the nature of the primary disease. Relapses are unhappily not infrequent.

Treatment.—The first essential in all cases is *rest*. Even in mild cases this should be made as absolute as possible for some days at

least, and if it is adopted early, severe cases may be often converted into slight ones. The patient should be confined to bed and all movements of the affected limbs should be restricted as far as possible, if necessary, by a long splint. This treatment is applicable not merely to recent cases; obstinate and protracted cases which have resisted all other treatment often yield readily to it. It is often necessary to continue it four to six weeks, or even longer, without break. Where it is not possible to obtain such complete rest, an effort should be at least made to avoid all movements which give pain and cause stretching of or pressure on the nerve.

Constitutional conditions, which are often predisposing causes, should receive adequate treatment. In some of the acute cases the salicylates relieve the symptoms; iron and arsenic often prove useful in cases with anemia. The rectum should be emptied and constipation avoided.

In acute stages the application of hot poultices along the course of the nerve may ease the symptoms. Counterirritation by the application of the cautery or by blisters over the course of the nerve is very generally employed, and often seems to be of distinct value. Baths, douches, and especially the hot-air bath, undoubtedly give relief and often influence the course very favorably, especially in its most chronic stages. When there is acute pain, symptomatic treatment may be forced into the first place. Antipyrine, phenacetin, and such drugs often give relief for a time. Occasionally deep injections of morphine or cocaine into the nerve may be necessary, but they are only temporary measures, and, owing to the nature of the disease, there is always a considerable danger of a drug habit. Morphine is the most effective, but cocaine in doses of from one-eighth to one-fourth of a grain may abolish all pain for hours.

Favorable results have been obtained by the injection of relatively large quantities of normal saline solution (50 to 100 c. c.) into the sheath of the nerve. The injection is made in the upper part of the thigh; severe pain referred peripheralward, paresthesia, and muscular spasm of the limb indicate when the needle enters the nerve. The acute pain disappears rapidly when the injection is commenced, which should be made slowly. More than two or three injections are rarely necessary, and often one is sufficient. Bum recorded the result of this in 73 cases; he obtained a complete cure in 42, and 14 were much improved.

Half a gramme of antipyrine dissolved in an equal weight of distilled water has been also recommended as a local injection into the nerve. Excellent results have been also obtained by the injection of 10 to 15 c. c. of physiological salt solution, or 1 per cent cocaine or 4 per cent stovaine, into the epidural space by Cathelin's method (Heile).

The galvanic current is often useful in the later stages; one large electrode should be placed over the nerve in the upper part of the thigh, the other on the leg over one of its branches or on the foot, and a constant current of 3 to 5 milliampères employed for 5 to 20 minutes.

It is rarely advisable to use massage in acute cases, but when the muscles become flabby or atrophied it is of service, but pressure or tension of the nerve must be carefully avoided. Acupuncture may be tried; a series of six or more sterilized needles are thrust in to a depth of about 2 inches along the course of the nerve in the upper half of the thigh and left there from 20 minutes to an hour. Many of the needles may pierce the nerve, but if they are inserted from above downward only the first one causes much pain.

Nerve stretching has also fallen out of fashion, and probably rightly, but it may be necessary to have recourse to it in very obstinate cases. Bardenheuer has suggested cutting away the brim of the sciatic notch where the nerve passes over it in order to leave the latter embedded in the soft tissues and free from pressure.

Diseases of the spinal roots.—The spinal roots may be affected by disease, either inside the dura mater or in their extrathecal course, before they anastomose to form the plexuses. The lesions of the latter portions have been considered under the plexus lesions and the diseases of the cauda equina are dealt with in another section. The disease of the roots may be primary or the symptoms may be caused by compression or extension of disease from the surrounding parts.

Primary root lesions are extremely rare. One root alone may be injured by a tumor in the neighborhood of the vertebral column, as frequently happens in malignant disease in this region, or spinal caries. Supernumerary ribs may produce an isolated palsy of the first thoracic nerve. But, as Dejerine and his pupils have pointed out, root lesions are more frequently secondary to intradural disease, and especially to compression and the invasion of them by local tuberculous or syphilitic meningitis. A single root, either sensory or motor, only may be affected, but more commonly several roots are involved. There are frequently signs of associated disease of the spinal cord.

The distinguishing feature of all root lesions is the limitation of the symptoms to disturbance of the functions of the motor or sensory fibers of the root or roots affected; this distinguishes root lesions from lesions of the peripheral nerves, which almost invariably contain fibers of two or more roots. The onset, which may be either acute or slow, is generally at first paroxysmal but may become continuous, is, as a rule, very severe and of the same darting or shooting character as the pains of *tabes dorsalis*. If it is the intradural portions of the

roots which are affected, sneezing or coughing may produce very acute pain in the affected root areas (*signe de l'éternuement*) owing to the effect of the sudden increase of the intradural pressure on the irritable fibers. After a variable time the pain gradually diminishes, and the skin which was previously hyperesthetic become hypesthetic as the sensory root fibers degenerate or are destroyed. When only one root is involved this diminution of sensibility may escape notice owing to the considerable overlap of the adjacent root fibers; it is largely dissociative in character, the loss of pain sensation being, in contrast to the condition found after peripheral nerve lesions, more extensive than the insensibility to light touch (Head). Simultaneously with the appearance of hypesthesia the muscles supplied by fibers from the affected ventral root or roots become paretic; and if the lesion is sufficiently intense they atrophy and changes in their electrical reactions develop; but as almost all muscles receive fibers from two or more roots the paralysis of any muscle is rarely complete if the lesion is uniradicular. Cutaneous trophic changes have been observed in the area of sensory distribution of the affected roots, probably in cases in which the root ganglia have been involved, and the ocular symptoms of irritation or paresis of the cervical sympathetic fibers may appear when the lower cervical and upper dorsal roots are affected.

Diagnosis.—This depends on the essentially radicular distribution of the symptoms. Spinal diseases, as *tabes dorsalis*, *syringomyelia*, and local lesions may produce symptoms of this distribution, but these can rarely offer any difficulty. From local neuritis of a peripheral nerve it may be also distinguished by the fact that the nerve trunks are never very tender to pressure. When the primary disease is a meningitis which invades or constricts the roots lumbar puncture may aid.

Treatment.—This should be directed to removing the cause; surgical intervention may be successful in the case of tumors, and if there are other symptoms of meningitis vigorous antisyphilitic treatment should be adopted if syphilitic infection can not be excluded. In cases with persistent pain the intradural section of the dorsal roots may be necessary.

PART 2.

ABSTRACTS FROM THE ENGLISH, GERMAN, AND FRENCH LITERATURE ON NERVE INJURIES IN WAR.

One law that can be laid down as a result of the study of the literature of war injuries of the nervous system is that the more peripheral the injury the more unanimous are the opinions of surgeons regarding the principles of adequate treatment. We have noticed the marked diversity of views in head surgery, as contrasted with the much less pronounced, though still plainly evident, differences of opinion regarding the treatment of spinal injuries. Regarding the peripheral nerve injuries, practically all authors are agreed that emergency operation is practically never indicated and that a leisurely careful study of symptoms is always in order; that it is often impossible to determine soon after the reception of the injury whether the nerve is totally divided, incompletely divided, or even whether it is in the slightest degree organically impaired. All are in pretty fair agreement, moreover, in insisting upon delicate operative manipulations, excision of scar tissue, and freeing of the nerve ends. It is also thoroughly agreed that much of the success following nerve suture depends upon proper after care. Most of the later articles recommend tubulization. On only one point does there seem to be much diversity of opinion, namely, final outcome after nerve suture; regarding this it must be said that too short a time has elapsed to judge the postoperative results accurately. One notes in passing, however, that one author reports complete restitution *two weeks* after nerve suture, whereas most operators expect little for from 6 to 24 months after operation. One author reports 75 per cent of good results and another one (who tubulized) reports failure to secure restoration of motion in a single one of 19 operated cases.

The physiological processes underlying nerve regeneration and nerve transplantation are so poorly understood that we have included as introductory abstracts the papers of Lewis and Kirk and Ingebrigtsen.

Kirk, E. G., and Lewis, D. D.: Regeneration in Peripheral Nerves; an Experimental Study. *Bull. Johns Hopkins Hosp.* 1917. xxviii, 71.

It has been shown by the authors that nerve defects may be bridged successfully by tubulizing with an autotransplant of fascia. Although devised primarily with reference to

practical surgical use, it soon became apparent to them that the method afforded unusual opportunities for a study of the histology of nerve regeneration following mechanical trauma. The defect was produced by excising a segment, varying in length from 1 to 3 centimeters, the nerve being cut squarely across with a sharp knife. Fascia lata from the same animal was used to construct the tube, since by using an autotransplant fibroblastic reaction and subsequent cicatrization were avoided. Thus regeneration following trauma could be studied without interference from various external factors and in particular the ingrowth of cicatricial tissue between the ends. Most important of all, the comparative behavior of proximal and distal stumps was more easily determined than when the ends were approximated.

The material used in the present report included 41 sciatic nerves of adult dogs, 21 of which were in complete serial section. The animals were killed at periods varying from one day to 36 weeks after operation.

The various methods of histological preparation for the study of the tissues are described by the authors in this paper. In conclusion they state that in the immediate vicinity of nerve trauma associated with break of continuity there occurs an accelerated hyperplasia of the neurilemmal elements which results in the early formation of protoplasmic bands, which develop in both the proximal and distal stumps and tend to bridge the defect. Along these protoplasmic pathways the regenerating axis cylinders from the central stump pass. Whether they reach the distal stump and neurotize, the authors state, depends largely on the extent to which these preformed conduits have prepared the way.

All efficient regeneration of nerve fibers (axis cylinders) is from the central stump, the authors believe, and all regenerating nerve fibers, whether the outgrowth of medullated or of nonmedullated axones, are in their early stages non-medullated.

They found that all medullation began proximally and proceeded distally, appearing only in those parts of the new axis cylinder which had acquired an age of five or five and one-half weeks (in the dog).

Lewis has described and illustrated the technique of fascial tubulization in *Surgery, Gynecology, and Obstetrics*, February, 1917, page 127.

Ingebrigtsen, R.: A Contribution to the Biology of Peripheral Nerves in Transplantation. *J. Exp. Med.*, 1915, xxii, 418.

From this experimental study the author draws the following conclusions:

Heteroplastic transplanted nerves become necrotic. They are unsuitable for bridges in cases of nerve defects, and his results explain the failure of the attempts at heteroplastic transplantation of nerves in human beings.

If it is desired to bridge a nerve defect by implantation, autoplasmic or homoplasmic grafts must be used. The occurrence of a Wallerian degeneration in these grafts during the first two or three weeks after the transplantation should make bridging a promising operation, for in this period the grafts resemble the peripheral part of a divided nerve and must be assumed to be capable of regeneration, and thus are very different from dead material.

The author has studied the process of regeneration and in a future article will communicate his results of bridging defects, which are encouraging so far as the function is concerned.

The contribution by Tubby is important as an attempt to formulate an exact definition of "nerve shock" (a condition similar to spinal shock) and the pathological changes incident thereto. It also emphasizes the importance of proper orthopedic measure and massage and electricity in the aftercare of nerve injuries.

Tubby, A. H.: Nerve Concussion Due to Bullet and Shell Wounds. *Brit. M. J.*, 1915, 1, 57. By Surg., Gynec. & Obst.

In reporting cases of nerve injury under his care at the Fourth London General Hospital, Tubby states that it is a little difficult to gather what is the general acceptance of the vague term "*concussion of nerve*." He thinks the following definition may prove acceptable: "*It is damage done to a nerve trunk without actual destruction of the axis cylinders; and the damage may consist of an effusion of blood between the fibers following compression of the nerve against a bone by the rapid passage of a foreign body in the immediate neighborhood of the nerve. In other cases the actual lesion may not amount to hemorrhage, but to a temporary anemia, or its opposite, hyperemia of the nerve, and specimens are required for microscopical examination before a precise diagnosis can be made. It is also possible to conceive that in certain large nerve trunks, such, for instance, as either of the popliteal nerves, where the motor fibers can be split up for a very long distance from the sensory, either a motor or a sensory bundle may be injured, so that in one case motor paralysis alone may exist and in another sensory symptoms be present.*"

In all cases stereoscopic skiagrams were taken. Where possible or practicable the shell fragment or bullet was removed, especially if it was near some large nerve trunk. Tubby says these physiological paralyses will clear up. A partial or irregular paralysis of muscles supplied by one nerve trunk is indicative of a physiological blocking such as arises from a small hemorrhage in or around a nerve trunk or a bruising. A persistence of the reaction of degeneration is an indication for exploration of the nerve. While waiting for the power to return he emphasizes the necessity of relaxing paralyzed muscles; e. g., wrist drop

to hyperextend on a splint, foot drop to dorsiflex the foot beyond a right angle. Massage and electricity should be given in these same position.

The following abstracts speak for themselves and require no critical comment.

Nonne, M.: War Injuries of Peripheral Nerves (Über Kreisverletzungen der peripheren Nerven). *Med. Klin., Berl.*, 1915, xi, 501.

The number of injuries to nerves is so great in the present war that, after they have been collected and compared, the knowledge of diagnosis and treatment in such cases will be greater than ever before. Not only are the numbers greater but the soldiers can be kept under observation and after-treatment administered better than in hospitals in time of peace. Nonne has found that the nerve is completely severed much more frequently than is usually supposed. Sometimes the severed ends are separated by as much as 4, 5, or 6 cm. Often the gap is filled in with cicatricial tissue or callus.

In cases where it is evident that the nerve is completely severed, operation should be performed early. If the nerve injury is complicated by fracture or other wounds, operation should be delayed till these are healed. But *in the majority of cases it is impossible to determine by neurological examination whether the nerve is severed*; the reaction of degeneration and disturbances of sensation and motility may be as great in cases of severe contusion or concussion. *In such cases there should be a delay of six or eight weeks to see if function improves without operation; if not, operate.*

The nature of the operation will depend on the condition of the nerve. Neurolysis is sufficient if the nerve is only strangulated or embedded in cicatricial tissue. If it is severed the ends should be freshened and sutured. If the ends are too far separated to be rejoined a piece of nerve may be grafted in. In taking hold of the nerves with forceps only the sensory fibers should be seized; an accurate knowledge of the topography of the cross section of the different nerves is necessary. Sometimes muscles react normally to the galvanic current and show the reaction of degeneration with the galvanic, and vice versa. Sometimes part of the muscles innervated by the nerve show the reaction of degeneration while others react normally; it is necessary to examine all the muscles carefully.

Attention is called to the frequency with which organic lesions are simulated by hysteria, and the author reports a number of cases in which he cured the paralysis following an injury by suggestion. He suspected hysteria because the tendon reflexes were normal. It may be necessary to anesthetize the patient to eliminate the hysterical element. After treatment in the form of electricity, massage, exercise, hot air, and hot water is of great importance in nerve injuries.

Hoffmann: Operations on the Peripheral Nerves (Unsere Erfahrungen mit der chirurgischen Behandlung der Schuessverletzungen peripheren Nerven). *Muenchen, med. Wehnschr.*, 1916, No. 34, Aug. 22.

The indications for surgical intervention are: (1) Complete motor paralysis with total reaction of degeneration; (2) partial motor paralysis, when after two or three months conditions remain unchanged or worse; (3) severe sensory irritative symptoms in the domain of the nerve which do not improve under treatment; (4) trophic disturbances, especially retardation of healing of wounds in the domain of the affected nerve.

The most suitable time for intervention is decided by the following indications:

(1) The wounds caused by gunshot and their complications must be quite cured, as a good result of nerve suture can not be hoped for unless in aseptic conditions. Even after apparent recovery germs may still be vital in a cicatrix, especially in fracture cases, and in such cases it may be necessary to delay intervention for eight or nine months until complete recovery and an aseptic condition is quite assured.

(2) Cases in which a grave nerve lesion is evident should be operated upon as soon as possible after recovery of the wound. Within two months 15 such cases were operated upon.

(3) All other cases should be operated on after a period of two to three months if there is no improvement in the nervous disturbances. Forty-seven such cases were operated upon two months or more after injury.

In technique the following points require attention: To approach the nerve with the least injury of soft parts; to respect the muscle nerve branches; rigorous hæmostasis using Esmarch's band; to proceed under general anesthesia.

In 58 of the cases the author practiced wrapping of the sutured tract or of the parts of the dissected nerve in a piece of free transplanted fascia lata taken from the thigh of the patient; it is necessary to include a large part of the aponeurosis in wrapping the nerve in order to avoid the following retraction which is always observed with nerve compression and its consequences. The wrapping fascis is fixed by suturing its margins together, and its extremities are sutured to the surrounding tissues. The author thinks that merely placing the nerve in the midst of muscular tissue is not the method of choice and refers to a case operated upon by others in which the sciatic nerve was so treated. The patient showed grave sensory irritative phenomena with advanced atrophy and flexional contracture. Having freed the nerve which he found adherent to the muscle, the author wrapped it in a fascial transplant and after three weeks the pain had disappeared and the patient could move the limb.

Of the end results, only a few particulars can be given owing to the necessity of clearing the patients. In 11 cases

of suture he had good results with return of function in periods varying from 2 to 12 months. In 7 cases the results were uncertain; 4 gave no results; and in 8 the time after operation is too short to give an opinion. In the 19 cases of nerve liberation, 14 were successful, 2 doubtful, 3 gave no results.

Ferrand, J.: *Neurology in War* (Réflexions médico-chirurgicales sur la pratique neurologique en temps de guerre). *J. de radiol.*, 1915, 1, 629.

Injuries of the large nerve-trunks are very frequent. The diagnosis is easily made, but the wounds rarely heal without suppuration, which renders operation on the nerve impossible. *Operations for the repair of nerves should never be undertaken within the first two months after injury, and sometimes even longer.*

When it comes to considering the question of operation there are three classes of cases:

1. Those in which the nerve is merely compressed by cicatricial tissue. Motor paralysis is not complete; there is not complete reaction of degeneration or vermicular contraction, but sometimes there is extreme pain. Operation is indicated in these cases and is very successful. The nerve is freed from scar tissue, displaced so that it runs through normal muscle, and the wound closed aseptically.

2. Those cases in which the nerve is partially severed. In these there is little or no pain and no trophic disturbance; paralysis corresponds only to the fibers that are severed. These cases should not be operated upon. The normal fibers serve as a guide along which the severed fibers are gradually reconstructed. Animal experimentation as well as clinical experience has shown that such reconstruction does take place. Electrotherapy is the sovereign treatment in this group.

3. Those in which the nerve is completely severed. In these cases motor paralysis is absolute in all the muscles innervated by the nerve in question. There is also anesthesia, complete reaction of degeneration in the peripheral end, and trophic disturbances begin to appear.

Opinions are divided as to the advisability of operation. Ferrand is inclined to think it is generally not indicated. When it is performed there is apt to be neuritis of the peripheral end, which interferes with regeneration. This is especially apt to occur in the painful cases, so that it is in these that operation is most contra-indicated. Some operators resect all scar tissue and resect the severed ends, but the author believes that such resection is rarely, if ever, successful; *he favors the more conservative method of simply dissociating the fibers from all fibrous tissue, leaving them to form a bridge for the reconstruction of new nerve-fibers.* Careful electrical examination is the most important point in making a differential diagnosis of the different classes of injury.

Thoele: Injuries of Peripheral Nerves in War (Kriegsverletzungen peripherer Nerven). *Beitr. z. klin. Chir.*, 1915, xcviil, 131.

Thoele devotes 125 pages to an exhaustive discussion of nerve injuries during the war. He has operated upon 46 cases. He found that in about half of his cases the nerve was completely severed, a higher percentage than is given by most authors. The radial was injured more frequently than any other single nerve. It was impossible to tell clinically whether the nerve was completely severed or not.

The results of 48 operations are reported; two patients were injured in two different regions and had two different operations performed. Neurolysis was performed in 17 cases, 9 simple and 8 complicated. In the 9 simple cases there were only two complete recoveries. In a third case the paralysis of the ulnar recovered, but with contracture of the third to the fifth fingers. In 3 more cases there was marked improvement which will eventually probably be complete recovery. There was slight improvement in 1 case and no improvement in 2. Of the 8 complicated cases there was slight improvement in 4 cases and very marked improvement in 4.

In 11 cases of complete severing of the nerve the ends were freshened and sutured. There was improvement in only 4 of these cases; complete recovery in none. In 10 cases the nerves were cut and sutured on account of spindle-shaped thickening; they were only partially severed. There is beginning motor improvement in 2 cases, and improvement in the electrical reaction in 2, though paralysis is still complete. The time is too short to judge of the other cases. In 5 cases the gap was bridged with flaps from the peripheral end of the wounded nerves. In 1 case there was only slight improvement. In 4 cases the fibers were separated and the injured ones sutured, with 2 positive and 2 negative results. In one case the peripheral end of the injured peroneal was implanted into the tibial which was also paralyzed from pressure by cicatricial tissue. There was no recovery of the motor power of the peroneal, though sensation was restored; the tibial recovered after neurolysis.

In general Thoele believes that when there is motor paralysis with partial reaction of degeneration it is best to wait six to eight weeks after the wound is healed in the hope that motility will be restored. In complete motor paralysis and complete reaction of degeneration operation should be performed as early as possible. Of course this may mean a delay of some weeks for the wound to heal, especially if there is suppuration. By early operation he means only as early as possible under aseptic conditions. This may mean from three weeks to three months after the injury. In partial paralysis with partial or complete reaction of degeneration it is best to wait 6 to 8 weeks after the healing of the wound. Operation should be performed as soon as possible in cases where there is extreme pain.

Operation should be done under general anesthesia. There is apt to be secondary hemorrhage after local anesthesia, and moreover, the electrical reaction of the nerve can not be tested during the operation if it is infiltrated with novocaine. He prefers not to cut off the circulation with the Esmarch bandage, because when it is applied vessels are overlooked, which cause secondary hemorrhage and lead to the formation of scar tissue again. The incision should be made in such a way that the skin sutures do not come over the nerve sutures. If this can not be avoided a flap of fat or muscle should be interposed over the nerve. Many surgeons make a sheath of fascia around the nerve, and various other substances have been proposed for these sheaths, such as calves' arteries, pieces of vein, rubber tubes, etc. Thoele believes that these sheaths contract and cause adhesions and strangulations of the nerve, so he thinks it is better to use only a flat flap of muscle or fat, not inclosing the nerve in a sheath. The different bundles of the nerve have different functions, so it is important to bring the corresponding bundles together in suturing. The nerve should be spared manipulation as much as possible. In seizing it with forceps only the nerve sheath should be grasped, not the nerve substance. When nerves have to be held aside it should be done with strips of gauze, not with instruments. The sutures should pass only through the epineurium. The stumps must be brought together without tension. Three or four button sutures are enough. Better adaptation is obtained with these than with a circular suture of the sheath. It is well to flood the nerve after suture with a 0.5 per cent novocaine-suprarenin solution. The limb should be placed in the best position to relieve the nerve from tension and it should be kept in a plaster cast for three to four weeks. When the gap is too great to admit of direct suture a flap is made from the injured nerve itself or from another sensory nerve. Where the nerve is only partially severed the author does not advocate complete section and suturing, but dissociation of the nerve fibers and suture only of the injured ones, leaving the uninjured ones intact.

The after treatment consists of electrical treatment with the galvanic current, for the faradic current has no effect; mechanical exercise; and local baths of various kinds. Neuralgia is not always overcome by neurolysis. Heile thinks that excision of the perineurium is of value in neuralgia. In severe cases of neuralgia in mixed nerves where neurolysis has not been effective Thoele resects the sensory tracts from the nerve trunk. In purely sensory nerves he resects and sutures if there is scar tissue, or resects a piece of the nerve and bridges the gap with a flap from the peripheral stump.

Wilms: Early Operation, Mechanics of Nerve Injuries and Technique of Suture (Zur Frueh-operation, Mechanik der Nervenverletzung und Technik der Naht). *Deutsche med. Wchnschr.*, 1915, xii, 1417.

Within the first 10 days to 2 weeks after an injury it is very easy to suture the divided ends of the nerve, and if some of the nerve fibers are intact to distinguish them from the injured ones and preserve them.

The conditions are very different in late operations. A large amount of scar tissue has formed between the ends of the nerve which must be removed, and a gap is thus left which requires great tension on the nerve to fill. The limb must be fixed for a long time in the position that removes tension from the nerve. Moreover bits of bone have frequently become incorporated in the scar tissue which could have been very easily removed at early operation.

The objection is urged against early operation in all cases that it is impossible to tell whether operation is necessary or not. Wilms proposes in all cases to make an exploratory incision to find out. This can readily be done under local anæsthesia, does no harm if unnecessary, and gives the patient much better chances for restoration of function if operation is necessary. The nerve fibers are generally displaced in the direction of the exit wound so that spontaneous restoration of function is improbable. To strengthen the suture it is well to leave a band of tissue from the external wall of the neuroma connecting the two ends of the severed nerve. Illustrations are given of how this is done. The sutured ends may also be enveloped in sheaths made of calves' arteries, fascia, or other material.

Delorme: Injuries of Nerves by Projectiles, Especially Injuries of the Sciatic (Sur les blessures des nerfs par les projectiles et en particulier sur les blessures de sciatique). *Rev. de chir.*, 1915, xxxiv, 402.

Delorme discussed the above subject before the Paris Surgical Society, basing his conclusions on a large number of cases that he had had occasion to operate upon. He is an advocate of operation after cicatrization of the wound, especially in cases where paralysis begins at once and does not show improvement. When the incision is made the ends are generally found several centimeters from each other, a large neuroma occupying the intervening space. In these cases he sections the nerve beyond the neuroma, brings the ends together and sutures them. In some cases there is cicatricial adhesion of the nerve to neighboring parts, but no break in the continuity of the nerve.

He explores carefully till he finds the limits of the lesion, excises the cicatricial tissue and sutures the freshened ends of the nerve together. In some cases of contusion of the sciatic that give rise to persistent pain, localized adhesions are found, but sometimes, even when the pain is intense, no visible lesion can be discovered.

In the discussion Gosset said that he had performed 60 operations for wounds of the peripheral nerves. He believes that every time an injury of a peripheral nerve is diagnosed an exploratory incision should be made, so that the lesion can be observed directly. This exploratory incision should be made within two or three weeks after the injury.

Routier expressed surprise at the number of operations performed by Delorme, and at the fact that he applied the same treatment to all cases. He has only operated in three cases.

Léguen pointed out that there are two objections to early operation in nerve injuries: Persistent suppuration and the difficulty of making an exact diagnosis of the nerve lesion. He advocates exploratory incision.

Delbet expressed surprise at the large number of operations performed by Delorme, and at the fact that he seemed to have sacrificed the nerve trunk without having any exact information as to its anatomical condition or its physiological value.

Quénu declared that it was impossible to recognize with the naked eye whether the nerve was intact and to determine the anatomical value of a nerve cicatrix.

Hezel, O.: Injuries of Peripheral Nerves During War (*Kriegsverletzungen des peripherischen Nervensystems*). *Med. Klin.*, Berl., 1914, No. 45, 1663.

From the experience derived during the last wars, it is evident that 1 to 2 per cent of all injuries are complicated by injuries of or damage to peripheral nerves. The peripheral nerves may be injured by gunshot wounds, stab wounds, crushing injuries, and by infectious toxins. Infectious neuritides arise from infected wounds. Most frequent injuries are the gunshot injuries, which may be direct and indirect. Not only the nerves struck directly by the bullet are injured, but others more distant from the bullet canal. A distant action still unexplained takes place here. The symptoms of the distantly injured nerves retrogress in time, whereas those symptoms due to direct injury of the nerve are more or less permanent unless operative measures are instituted and the nerve sutured. Examination does not reveal whether in a groin case of nerve injury a complete severance of the continuity of the nerve or only a complete functional inhibition with a retained continuity exists.

In cases of nerve injury by blunt force without a penetrating wound, even in the presence of complete functional inhibition, a restoration of function is much more probable than in injuries by bullets. Operative interference is not at all considered in such cases. In stab wound injuries of peripheral nerves, it is possible only in the rarest of cases to obtain functional conduction without surgical interference. As a rule Hezel recommends that

operations on the nerves be performed as soon as the necessity of such an operation is apparent, provided the wound conditions permit. Not only motor disturbances, but also neuralgias at times are indications for surgical interference.

Edinger, L.: The Uniting of Divided Nerves (Ueber die vereinigung getrennter Nerven. Grundsatzliches und Mitteilung eines neuen verfahrens). *Muenchen med. Wehnschr.*, 1916, lxiii, 225.

Edinger has found that there is often great difficulty in the union of the ends of severed nerves. The regenerated nerve-fibers which are thrown out by the ganglion cells can easily be diverted from their course by any mechanical obstruction, such as a blood clot, and union between the stumps can therefore be prevented. He shows that this is the case by his own observation and those of others whom he quotes.

The only way that the regenerated fibers may be kept in the proper direction to effect union is to permit them to grow in a tube. Nevertheless the attempts made to grow nerve fibers in tubes by previous workers did not give good results because it was necessary for the fibers to be surrounded in the tube by a suitable environment for growth. The various experiments of Edinger demonstrated that *human nerve fibers grow best when the two disunited ends are inserted in an artery filled with agar jelly.* This is the new procedure which he advocates. A number of such tubes have been prepared and distributed for use to operating neurologists.

Edinger has seen the results obtained by Ludloff and Hasslauer with 14 patients treated in this manner, in which cases the distance between the disunited nerve ends varied from 5 to 15 cm. In every case there was clear evidence of good progress of regeneration in the nerve. Within a few weeks the anæsthesia area became much reduced. He mentions particularly a case in which 10 cm. of the tibial and 8 cm. of the popliteal nerve had been resected. After inserting the agar jelly tube the return of the plantar reflexes was demonstrable after 16 days.

Borchardt, M.: Gunshot Injuries of Peripheral Nerves (Schussverletzungen peripherer Nerven). *Beitr. z. klin. Chir.*, 1915, xcvi, 233.

As consulting surgeon of the third army corps and surgeon of the military prisoners at Zossen, Borchardt has seen several hundred cases of nerve injury, and has operated upon more than 70. In this article he gives the history of 56 cases. Of the series, 17 were injuries of the radial, 8 of the median, 7 of the ulnar, 7 of the ulnar and median, 1 of the musculocutaneous, 8 of the brachial, cervical, or lumbar plexus, 8 of the sciatic. Excellent plates are given, containing 39 illustrations showing the operations.

The indications for operation were decided upon according to the principles laid down by Oppenheim, Cassirer, and other noted neurologists. *Operation was performed when the neurological findings indicated that there was severe injury of the nerve, either from partial or total section of the nerve or from scar formation around it. He operated if there was complete motor paralysis, complete reaction of degeneration, or severe disturbances of sensation.* Operating on these indications, he met with negative findings on operation in only 2 cases. Among the 56 cases, in 18 the nerves were found completely and in 1 partially severed. The stumps were generally swollen, with club-shaped ends. In the other cases there were more or less extensive scars, and sometimes foreign bodies were embedded in the nerves, such as bits of metal or cloth, fragments of bone or muscle fibers.

In operation the greatest care was taken to control hemorrhage, *so as to avoid the formation of even the smallest hæmatomata*, which might lead to renewed scar formation. If the tissue around the nerve showed cicatricial changes, a sheath of fat or soft fascia was put around the nerve. The chief factors in the early restoration of function are careful suture, absolute asepsis in operation, the avoidance of hæmatomata and early movement, massage, and electrical treatment.

Direct suture is to be preferred to all other methods. Care must be taken in suturing to bring the corresponding nerve tracts into exact apposition. The muscles should be spared all injury so far as possible. If palpation or exploratory incision shows cicatricial tissue, it should be excised. Of course, the ideal procedure would be to excise into sound nerve tissue, but the histological examination of apparently normal cross sections of nerves gave surprising results. The plates show some histological pictures of cross section of nerves were apparently normal, but the microscope reveals the fact that they contain considerable cicatricial tissue, and in some places there are no nerve fibers left at all, or else they are only tubes of nerve tissue filled with scar tissue. But Borchardt believes, as does Cassirer, that it is better to suture such nerves, even though they contain some scar tissue, than to remove such large pieces that they can not be sutured together. If only a part of the nerve is injured, electrical examination will show what part of it should be resected.

Borchardt recommends that operation be done as early as possible, because it is technically easier then and because early operation prevents contractures of the muscles and joints and trophic disturbances. In the author's own cases the time between the injury and the operation varied from two weeks to nine months. In 6 cases there were also injuries of the vessels, but the vessel wounds closed spontaneously, without the formation of aneurisms. Among the 56 cases, 25 were under observation longer than three months; 21 of these were improved and 4 not improved. The results

were particularly good in 3 cases of nerve suture, function being completely restored. *Borchardt believes that the results of operation have been given more praise than they deserved.*

Heinemann, O.: Gunshot Injuries of the Peripheral Nerves; Anatomic Investigation of the Inner Structure of the Great Nerve Trunks (Ueber Schussverletzungen der peripheren Nerven; anatomischen Untersuchungen ueber den inneren Bau der grossen Nervenstaemme). *Arch. f. klin. Chir.*, 1916, cviii, 107.

Heinemann finds that nerve suture has in general given 70 to 80 per cent positive results. *In his own cases he obtained 75 per cent good results.*

Although Stoffel has condemned nerve transplantations, yet by this means Gratyl obtained 66 per cent successes in nerve defects. The prognosis of gunshot injuries of the nerves is in Heinemann's experience good. Previous to the war it was known that the reconstitution of nerve functioning took a very long time. *It takes about two years before it can be stated with certainty that there is no return of nerve functioning.* Heinemann's optimism is based upon his observations of recovery in apparently noncurable cases. There were only two cases of nerve suture in which positive results were not obtained and these are still under observation.

The most striking successes are obtained in neurolysis. Paralysis may disappear within 24 hours; whereas after resection such a result is not usually obtained till after two months.

For the aftertreatment of nerve injuries Heinemann advocates electricity. Systematic electrical treatment greatly facilitates recovery. This is particularly the case in patients with weak will power.

Tuffier, T.: Treatment of Injuries of the Nerves by Projectiles (Traitement des lésions des nerfs par projectiles de guerre). *Bull. et mém. Soc. de chir. de Par.*, 1915, xli, 1911.

Tuffier reported the work of Dumas on 280 cases of nerve lesions during the war. From his results he concludes that section and suture of the nerves should be practiced only when no other treatment is possible. Among his 280 cases, *nerve suture was practiced in only 19, and in none of these cases was motion restored.*

The treatment of choice is to liberate the nerve from scar tissue and make a sheath for it of some substance that will protect it from further cicatrization. The nerve should be handled as little and as gently as possible, and the cicatricial tissue developed within the nerve itself should not be touched. Even where the nerve has been completely severed the conservative method is still indicated.

Dumas reports a case to illustrate his method of leaving a bridge of sclerotic tissue about the size and shape of the nerve between the severed ends. Gradually nerve fibers grow

through this from the distal to the peripheral end and function is restored. After the nerve is freed it must be protected, and he finds that the best material for this purpose is fatty tissue. This may be taken from the patient himself or from some one else. Its softness and elasticity make it an ideal material for protecting the nerve from compression by new-formed scar tissue. Other materials frequently used, such as hernial sac, veins, and aponeurosis, are not thick enough to give as efficient protection.

He has found this method particularly effective in those very painful lesions of the median nerve which many operators reported as helpless.

Lyle, H. M. M.: *The Physiological Treatment of Bullet and Shell Wounds of the Peripheral Nerve Trunks.* *Surg. Gyn. and Obst.*, 1916, xxii, 127.

Lyle, from his experience in the war zone, draws the following conclusions:

1. Damage to an important peripheral nerve is an injury of extreme gravity.
2. Primary nerve suture is rarely indicated.
3. Unrelieved, overstretched muscular tissue leads to fatty degeneration and loss of contractility.
4. A paralytic deformity with shortened muscle and limited joint movement, in the majority of cases, is the result of ignorance or neglect.
5. It is imperative, whether the nerve is divided or not, that the paralyzed muscles be relaxed and protected from strain by a suitable apparatus. Under no circumstances must this be deferred to the so-called after treatment. The postural prophylaxis begins with the reception of the wound, and continues after the operation until voluntary motion is restored. A strict adherence to this vital orthopedic principle aids in the diagnosis, hastens recovery, prevents many distressing deformities, and will materially diminish the number of useless limbs.

Marburg, O., and Ranzi, E.: *Gunshot Injuries of Peripheral Nerves* (Zur Frage der Schussverletzungen der peripheren Nerven). *Wein. klin. Wchnschr.*, 1915, xxviii, 611.

From experience with 2 nonoperative and 48 operative cases of nerve injuries the authors come to the following conclusions:

1. When after a gunshot injury there is loss of motion and sensation and complete lack of electrical reaction, operation is indicated as soon as the wound has healed.
2. When there is loss of motion and sensation and the electrical reaction is growing worse, operation is indicated.
3. When there is loss of sensation and motion, with no tendency to improvement, and the reaction of degeneration remains stationary for several weeks, operation is indicated.
4. If there are suppurating wounds, operation should be delayed for several weeks.

Stoney, R. T.: Nerve-Suture for Bullet Wounds. *Brit. M. J.*, 1915, ii, 10.

As an operating surgeon in the French Army the author had many opportunities of seeing cases of nerve injury caused by modern weapons. From four operated cases he concludes as follows:

1. The function of a nerve may be interrupted without material injury, in which case the loss of function is only partial and returns early, probably within a fortnight or three weeks.

2. When a nerve is partially or wholly divided loss of function is marked and permanent and may even lead to increase. In these cases it is useless to expect spontaneous regeneration, owing to the distortion and separation of the cut ends and the great development of dense fibrous tissue, which appears to follow in all cases.

3. When a nerve is divided, the sooner an operation for its suture is performed the easier it is and the greater the likelihood of an early cure. In cases, however, where the wound is septic, it may be advisable to allow time for the wound to heal.

4. Even when no treatment has been given for several months there is still a chance of a successful result if late suturing is undertaken, so that no case need be looked upon as necessarily hopeless.

Auerbach, S.: Treatment of Gunshot Injuries of Peripheral Nerves (Zur Behandlung der Schussverletzungen peripherischen Nerven). *Deutsche med. Wchnschr.*, 1915, xli, 254.

There is a great deal of difference of opinion as to whether gunshot injuries of the peripheral nerves should be treated operatively or conservatively and as to how long electrical and mechanical treatment should be continued before operation is undertaken. From his experience thus far Auerbach is inclined to adopt the following rules:

1. Those cases are to be treated conservatively in which the motor and sensory disturbances are slight and in which electrical examination reveals only a slight decrease in electrical excitability or a partial reaction of degeneration. In such cases there is an improvement in function in three or four weeks, although complete recovery may take eight weeks or even three months.

2. Those cases should be operated on in which there is complete motor paralysis and complete reaction of degeneration. As soon as the wound is healed the nerve should be laid bare and its condition determined and the operative indications decided upon. Neurolysis may be performed, embedding the nerve in sound muscle tissue, or the nerve may be inclosed in tubes of various materials, or if the nerve trunk is completely severed nerve suture may be done. If there is extensive loss of substance of the injured nerve, one of the various plastic operations on nerves may be performed. If there is a neuroma, the nerve should be resected

into sound tissue and a plastic operation performed. If there are callous changes, such segments of the nerve should be resected.

3. It is more difficult to decide on treatment in the transition cases between the first and second group, but *Auerbach is inclined in doubtful cases to advise exposing the nerve*, as it is not a dangerous procedure. If conservative treatment is preferred, he would advise that if there is no functional improvement in six or eight weeks operation should then be performed.

4. Operation is also indicated in cases in which there is severe and long-continued pain. This complication is quite frequent. Of course, operative treatment in all cases must be followed by systematic electrical and mechanical treat-

Cassirer, R.: Operative Treatment of Injuries of the Peripheral Nerves in War (Die operative Behandlung der Kriegsverletzungen der peripherischen Nerven). *Deutsche med. Wchschr.*, 1915, xli, 520.

Cassirer gives histories of three cases. The first was paralysis of the radial from a fragment of a shell. Operation was performed two weeks after the injury; the nerve, which had been severed, was sutured. Three and one-half months after operation there were signs of returning motility, which slowly but steadily progressed. The second had paralysis of the deep branch of the radial. Four weeks later the nerve, which was completely severed, was sutured; eight weeks after the operation there was movement in the paralyzed region, which increased rapidly in strength and extent. The third case was a fracture of the humerus, with injury of the radial, followed immediately by paralysis. Operation was performed three months later, consisting of neurolysis and extirpation of a piece of bone from the nerve. After six weeks improvement began.

The author has seen about 240 cases of nerve injury, in 60 of which operation was indicated. In over 25 per cent of these the nerve was completely severed; in the other 180 neurological examination showed that operation was not indicated. There was no reaction of degeneration and motor and sensory functions were preserved. Expectant treatment is generally advocated in nerve injuries, but Cassirer thinks that in all cases where neurological examination indicates operation it should be performed promptly as soon as the wound is healed. He thinks the advantages of early operation far outweigh its dangers.

Chiray, M., and Roger, E.: Nerve Sutures (Des sutures nerveuses). *Bull. et mém. Soc. méd. d. hôp. de Par.*, 1916, xl, 2149.

The authors in a long and exhaustive article point out that there is need for unanimity among neurologists regarding the classification of cases to be included in statistics of nerve suture; also as regards the criteria of restoration. They

define four classes of motor restoration: (1) Lesions with no motor restoration; (2) lesions with slight motor restoration; (3) lesions with advanced motor restoration; (4) lesions with complete motor restoration. The third class includes a return of voluntary motility sufficient for the execution of movements of the paralyzed muscle with the amplitude but without the normal strength. The fourth class includes complete restoration of amplitude and motor force.

From their extensive investigations into the results of nerve suturing the authors conclude:

1. Cases for suture should be selected with care and suture confined to cases of total and complete section of the nerve. As regards restorations, it is necessary to know what errors can arise in observation, as thereby false conclusions may be reached regarding motor or electrical restoration.

2. From chronological reports of the different stages of electrical and motor restorations, the authors find that the first always precedes. The beginning of the reappearance of movement is, according to their experience, in about five months for the radial, eight months for the cubital, seven months for the median, two to five months for the popliteal sciatic. The radial and popliteal sciatic nerves give the best results.

3. According to the authors' experience the result is the more favorable according as intervention is early. But even so, operations done from the fourth to the sixth month after injury give a large proportion of successful results.

The important points in every intervention are the total resection of all fibrous tissue, the necessity of coapting without torsion, without dragging, and without crushing the nerve. End-to-end suture and grafting give equally good results.

4. The authors are convinced of the importance of post-operative care, particularly of the functional prosthesis and ionization with iodide of potassium about the operative cicatrix and the nerve suture.

Heile and Hezel: Experiences in the Treatment of Peripheral Nerves Wounded in War (Unsere bisherigen Erfahrungen bei der Behandlung im Kriege verletzter peripherer Nerven). *Beitr. z. klin. Chir.*, 1915, xcvi, 299.

The scarcity of dependable data concerning the handling of wounds of peripheral nerves in previous wars and the extraordinary number of cases which have presented themselves in this war have led Heile and Hezel to report in detail the neurologic findings and operative procedures of 40 cases. It is their intention to report later concerning the results obtained.

Heile discusses the surgical procedures. He considers operative interference desirable if no improvement has occurred in from four to six weeks after the injury was sustained. A general anesthetic is to be preferred, not only because such operations require a long time, but because the

hemorrhage which supervenes after a local anesthetic is likely to interfere with the growth of the sutured nerves.

In the majority of cases the nerve trunk is not completely severed. It is of great importance to avoid injuring such unbroken fibers whenever possible. An attempt was made in some cases to search out the corresponding bundles in the proximal and distal ends and to suture them, but the difficulties were very great. Much time and care is required to dissect the nerve trunk out of the scar tissue in which it is usually embedded. This may be facilitated by beginning at either side of the scar and loosening the nerve for a short distance in the healthy tissue, holding it up by thin strips of gauze and by gentle traction, putting the adherent portions on the stretch. The nerve sheath is then split and loosened from the nerve trunk. In the healthy portion this is easily accomplished with a blunt instrument, a small elevator, or strabismus hook. By the injection of air or salt solution the sheath is ballooned out and loosened from the trunk. Over the injured portion the perineurium may be markedly thickened and pressing on the nerve. In such a case a sharp instrument is required to loosen it. If neighboring bones are broken, there may be splinters of bone in the scar or even in the nerve, or the callus or bony spines may be pressing on the nerve. The separation of the very firmly adherent blood vessels is very difficult and often further complicated by injuries to the vessel walls. These aneurismal enlargements often can not be diagnosed in advance on account of the intervening scar tissue.

When the proportion of broken to unbroken bundles is small it is not so difficult to adapt the distal and proximal ends of the fibers which belong together, but when the proportion is reversed this is frequently not possible. A little help may be obtained by laying the fibers in their apparent anatomical arrangement before suturing. The motor and sensory fibers may be distinguished by electricity, but this can not always be used, as in the majority of cases the distal portion can not be stimulated by either the galvanic or the faradic current, and in others the proximal portion may fail to be stimulated. Electricity is, however, useful at the beginning of operation in badly distorted cases to distinguish the principal nerve trunks, as the median from the ulnar, etc. It is hopeless to try to associate by this means the central and peripheral portions of individual fibers. Experience in former wars seems to show that such careful adaptation is not of great importance. Whenever the whole nerve was severed or severely injured, the necessary resection was done and the ends sutured in the best way to avoid stretching if possible. For suture material fine silk was used at first; later fine catgut. Whenever individual nerve-fiber bundles remained intact they were used as splints for the sutured ones. Unless tension made it necessary to go deeper the stitches have included only the supporting substance of the nerve, but it is always necessary to see to it that the portions brought into contact consist of pure nerve substance.

Whenever the perineurium was sufficiently thickened to press upon the nerve, it was removed as a foreign body. It was also frequently removed in cases in which it merely showed definite symptoms of inflammation, and especially in cases which showed symptoms of peripheral neuritis. In many cases the pain was permanently relieved in this way, in others it returned after a while, but these latter were apparently cases of ascending neuritis. The sheath should, in any event, be split lengthwise to free the nerve bundles of the inflammatory exudate between them. Such an exudate may result from the suturing of the nerve. Therefore, the sheath should be split for several centimeters on both sides of the suture, and this slit should not be resutured.

In cases requiring resection up to 6 centimeters, the central and peripheral ends of the nerve were dissected out of the soft parts and displaced subcutaneously as far as possible; the distance was decreased by flexion or extension, and finally, by fine spiral incisions in the perineurium, the ends were lengthened somewhat. Stay sutures along the sides of the nerve were used to assist in holding the approximated ends together, and if the tension was great, these stitches had to include nerve bundles to avoid tearing out. Great care was exercised to see that nothing was interposed between the active nerve substance of the sutured ends. Finally, it is necessary to protect the sutured nerves from pressure, especially in cases of bone fracture. This is best accomplished by the interposition of a neighboring muscle, or a pedunculated muscle flap.

In cases in which it was necessary to use tubes, rubber tubes, prepared from pure rubber and not vulcanized, were used. The tubing was boiled in salt solution and split lengthwise. Prepared in this way it can be used to inclose the stumps of nerves or it can be used to protect the sutured nerve from its surroundings.

Hezel describes the 40 cases in detail, giving the point of entrance and exit of the bullet, which nerves were injured and how badly, a description of the findings upon operative exposure of the part, and the surgical procedures applied. The neurologic examination included, with a few exceptions, only the motor functions. The injuries were classified as severe, moderate, and light. In severe cases, the nerves were not responsive to either the galvanic or faradic current, and the muscles did not respond to the faradic and but sluggishly to the galvanic. In moderate cases the electrical irritability of the nerves was not absent, but materially reduced quantitatively, and sometimes altered qualitatively; the muscles qualitatively. Light cases showed at most quantitative reduction, no qualitative changes. The findings upon exposure of the injured area vary according to whether or not the nerve is completely severed. If it is completely severed, both the ends are usually embedded in dense scar tissue with a space between them. Unless the operation is undertaken very early, the central stump will show a swell-

ing consisting of a neuroma. Otherwise the severed nerves are not much enlarged, and the peripheral portion may even be somewhat atrophied. If the nerve is not broken, but merely grazed or crushed by the shot, there will be an irregular swelling of several centimeters length distal to the point of injury. This is doubtless caused by inflammatory exudate inside the nerve-sheath with consequent obstruction of the venules and lymphatics of the nerve. This swelling, which may be twice or even three times the diameter of the nerve, is gradually reduced, and induration of the nerve-sheath and interstitial tissue takes the place of the infiltration. In cases in which the nerve is penetrated by the shot, so that the sheath is opened, this distal swelling is entirely absent, and the nerve on both sides of the lesion is slightly swollen, soft, and reddened. Upon opening the sheath of a nerve that was not cut by the shot, one frequently finds more or less of the contained fibers ruptured with scar connective tissue between the ends of the fibers, and if sufficiently late, the beginning development of neuromata. These individual fibers, even as the whole nerve under similar circumstances, must be resected and the ends freshened before regeneration is possible.

There is as yet no diagnostic method of determining whether or not in severe cases there is destruction of continuity of the whole nerve or only of some of its fibers. Neurologic examination will show disturbance or absence of functions, and in every case of absence of conductivity the possibility of loss of continuity must be considered.

Kaiser, F. J.: *Neuralgia After Gunshot Injuries* (Ueber Neuralgien nach Schussverletzungen). *Beitr. z. klin. Chir.*, 1915, xcvi, 256.

Kaiser gives the histories of six cases of neuralgia after gunshot injuries of the limbs. Four of the cases were in the median and two in the sciatic. In all of the cases except one there was a mixture of neuralgia and neuritis. One was a pure neuralgia. The neuralgia generally begins a few days after the injury and proceeds slowly without any thickening of the nerve and without any trophic disturbances of the skin. The neuritis begins immediately after the injury and disappears sooner than the neuralgia. The inflammation is generally only perineuritic and does not cause interruption and destruction of conducting nerve fibers.

The treatment is tedious and consists of hot air, hot baths, massage, and electricity. Good results were obtained from the injection of 1 per cent novocaine and adrenalin, followed by massage; injection of fibrolysin into the scar was also of value. When these methods were not effective (two cases) the nerve was laid bare, freed from cicatricial tissue, and sheathed in a flap of muscle. Stoffel has recently published an article showing the positions of the limbs in which the nerves are under tension and in which they are relaxed. In these cases the limbs were held in the characteristic positions for relaxation of the nerves.

Borchardt: Report on Gunshot Injuries of Nerves (Ueber Schüsse insbesondere Spätkirurgie). Report to the Second German Surgical Congress. *Beitrag zur klin. Chir., Bd. V, Hft. 1, Kregs Chirurgische Hefte*, 1916.

Borchardt considers the treatment of gunshot injuries of the peripheral nerves. The indications for operation he states as follows:

Every case of severe nerve injury should be operated, and operation should be done upon the grounds of findings, not upon the course of the recovery. The symptom complex which he characterizes as distinguishing severe injury, is the following: Total failure of function of the nerve, motion and sensation gone, trophic and vasomotor disturbance, and complete reaction of degeneration. If there is only partial reaction of degeneration, he does not operate, only when there is no longer any improvement or where there is decided symptom increase. When there is complete loss of function of the nerve, it is impossible to distinguish whether the nerve is completely divided or whether there is simply a lesion interfering with the conductivity of the nerve, and for the surgeon it is not necessary to distinguish. If the case is one of severe injury in the above sense, it should be operated as soon as the condition of the wound permits. *One can not determine by electrical examination whether the nerve is torn or not.* It is often quite difficult to make a differential diagnosis between psychic paralysis and actual nerve lesion. In these cases electrical examination is of highest worth.

Borchardt believes in early operation, and he cites one case which he operated 14 days after the injury with good results, but on the average he recommends the sixth to the eighth week as the proper time, because earlier than this the symptom complex is not distinctly developed, and the condition of the wound seldom permits observation. From a purely technical standpoint everything argues for early operation. The earlier the operation is done, the easier is the operation, because dense, extensive scars have not yet formed, and it is much easier to loosen the nerve than at a later date. The longer the nerve is compressed, the longer foreign body lies in the nerve trunk, so much worse is the prognosis. The longer a nerve remains nonconductive the longer it will require for a resumption of its physiological function. Further than this, early operation will in a measure avoid contractions and atrophies in muscles, bones, and joints, which are bound to occur if operation is delayed very long.

How long shall one wait for a possible spontaneous recovery of function? This is a difficult question. One case was seen in which one and one-half years after the injury the first signs of regeneration were observed. Borchardt is well aware that an early operation may interfere with beginning spontaneous regeneration, but, all in all, he believes that the sixth to the eighth week is the proper time.

He agrees with Wilms, in so far as recommending the operation upon exploratory grounds, but he has found in somewhat more than half the cases that nerve suture or other nerve operations are necessary. On this account he intimates that nerve injuries should be collected under the treatment of men who have given the subject special study.

Borchardt does not believe that primary union of a divided nerve is possible, and believes that in any case there is a degeneration of the peripheral portion of the nerve and that regeneration occurs by the axis cylinder growing toward the periphery from the site of division.

Borchardt operates in a bloodless field, secured by constricting bandage, because one must be able to recognize the anatomy most accurately. If the nerve is divided, the ends should be freshened sufficiently to remove all scar tissue, and the nerve should be sutured. The direct suture is the ideal operation to secure conductivity of the nerve. The finest silk sutures are passed through the perineurium and corresponding bundles are brought into approximation. He relies upon the microscopic relationships. In the majority of cases direct suture is possible. In cases of large defects it is sometimes possible by making large exposure of the central and peripheral stumps to stretch them sufficiently to bring them together. In some cases he has even left some scar tissue if there appeared to be slight tension upon the sutures. When the gap is too large to allow the ends to be brought together, then Borchardt prefers implanting the two ends into a neighboring nerve. He is not inclined to bridge the gap with foreign material. He favors inclosing the suture with material to prevent adhesions, when possible.

The period after which regeneration begins to manifest itself varies, and possibly not before one or two years can one tell whether regeneration will be complete. Several factors interfere with good results. The most common is that resection of the ends is not sufficient, scar tissue remaining upon the central and the peripheral stumps. Another possibility is that during the after treatment brisk movement causes separation of the sutured ends on account of tension. Third, is the compression of the nerve by adhesions and portion of scar tissue.

In all these cases a renewed operation is certainly indicated, but one should not repeat the operation too early. In this, all are agreed. In no case should one reoperate unless there is absolutely no sign of regeneration for a half year after the primary operation. In general, it is much better to attempt first to resuture the nerve before undertaking muscle or tendon transplantation.

Ernst Müller: Utilization of the Elasticity of Nerves, etc. (Ueber die Ausnutzung der Dehnbarkeit des Nerven Durch Temporäre Verkoppelung bei Großen Defekten zum Zweck der Nerven-naht). *Beiträge zur Klinischen Chirurgie.* (Kriegschirurgisches Heft XXX-XXXIV.) April, 1917, p. 651.

Müller has encountered several cases where there was a considerable gap between the nerve ends to be approximated, so that direct suture could not be carried out. In such cases, several procedures have come to be recognized. These he considers in the following order:

(1) Implantation, in which the nerve ends are implanted into a neighboring nerve. In some instances, instead of the implantation of the peripheral stumps into another motor trunk, both stumps are implanted into the same nerve trunk. (2) Interposition of nerve substance may also be done by interposing a flap from the peripheral stump. Free transplantation of nerve tissue may also be done and interposition of foreign material, such as catgut, has also been done. (3) Regeneration can also be accomplished by inclosing the two stumps, either in empty tubes, or filled tubes. The tube may be of decalcified bone, rubber dam, magnesium, or calves' arteries hardened in formalin. Tubes of these same materials may be filled with blood vessels, with serum, with gelatin, with blood, or agar. In certain instances, tubes have been made of fascia and of fat.

Müller has devised the following method for approximating the separated nerve ends: The limb is so flexed as to relax the nerve to the greatest possible degree and the nerve ends are approximated as closely as possible and held in this position by means of a strip or tube of fascia. After wound healing has been completed and the nerve ends fastened by the fascia, the limb is then gradually extended, even over-extended. The nerve trunk possesses a certain degree of elasticity and lengthens owing to the stretching, and at a second operation, by forcibly flexing the limb, very often the nerve ends can be directly sutured.

von Lorentz: Nerve Injuries and Their Treatment (Nervenverletzungen und Deren Behandlung). *Beiträge zur Klinischen Chirurgie Vierter Kriegschirurgischer Band.* (Kriegschirurgisches Heft XVI.) July, 1916, p. 248.

It has been estimated that $1\frac{1}{2}$ per cent of the injuries produced in modern warfare are complicated by injuries of the peripheral nerves. It would be well for the surgeon on the front to remember this and in applying first dressings to consider the immediate and the late treatment of nerve injuries. Many of the injured themselves recognize that a certain part of the body is paralyzed, but the well-trained surgeon will always think of the possibility of nerve injury and conduct his examination accordingly.

When nerve injuries are recognized, the dressings and splints must be so applied as to render subsequent treatment for a nerve injury most favorable. It often happens, through neglect of this point, that limbs are left in such position that the nerve ends purposelessly are separated by 5 or 6 cm., and, further, that joints are allowed to ankylose in the most useless position—for instance, the median nerve forms a bend in the elbow and at the hand and finger joints. An injury of the musculo-spiral should call for bending of the elbow and the overextension of the wrist joint. In no case should the paralyzed muscles be allowed to be rendered useless by overextension.

Great difficulty, even with the most accurate neurological examination, may be experienced in attempting accurate localization of the lesion. The electrical reaction renders great assistance, especially when there is complete division of the nerve trunk. When the lesion is not complete, the picture is more complicated.

As to the question of operation, Lorenz has adopted the rule in all cases, even in light injuries which do not show a tendency to improve within three or four months, to make an exploratory operation. Also, where the progress has been satisfactory for three or four months and then there is recrudescence of symptoms, and also where there is a distinct lessening of electrical excitability, operation is indicated. Another indication for operation is severe neuralgias, which are limited to the distinct nerve distribution.

It should be emphasized that no nerve operation should be undertaken until the wound is completely healed. To operate through granulating tissues is to court disaster. Even completely healed wounds may often harbor bacteria (latent infection), which become virulent after operation.

Lorenz advises against the application of Esmarch bandage during operation. He objects upon several grounds: (1) That the nerve may be actually injured by constrictor long applied; (2) that the nerve conduction may be interfered with during operation, and on this account the conductivity of the nerve can not be ascertained during operation; and, finally, that the blood supply, being cut off during operation, an unnoticed hemorrhage may occur, with hematoma formation after the wound is closed.

The question of anesthesia is not usually easily decided. These operations are usually tedious and prolonged. On this account, general anesthesia has a distinct advantage, while local anesthesia usually will not last for an operation of long duration.

The character of operation depends, naturally, upon the character of injury. It should be generally advised, however, that the operation be quite radical. The incision should be sufficient to permit of good exposure of the nerve, not only at the site of injury but for several centimeters above and below. It is often also necessary in order to permit mobilization of the nerve ends. It will be often necessary,

on account of scar tissue or callus, to resect the nerve ends, or the nerve ends may be found separated. The approximation of the nerve stumps should always be possible after good mobilization. Certain positions favor this by decreasing the tension of the nerve trunk. The suture should include just perineurium, and care should be taken to avoid rotation of the stumps, thus insuring direct contact of the corresponding bundles.

Subsequent adhesions at the site of suture should be guarded against in one way or another. Several methods are recommended for this trouble, the most rational being the inclosing of the suture line with an autoplasmic fat transplant.

One method of approximating nerve ends that are widely separated is that of resection of a piece of bone. This is hardly to be recommended in every case. In large nerve trunks direct suture may be possible through plastic operations, similar to tendon lengthenings. The old method of nerve implantation (i. e., the implantation of a peripheral nerve stump into another peripheral motor nerve), may still be employed, often with good results.

Primary healing of nerve suture is a very important consideration in the result. A correspondingly careful after-treatment is also of the greatest importance. Care should be taken to avoid tension on the nerve ends by keeping the joints flexed in the most suitable position, and dressings should remain in place until healing is complete. The paralyzed muscles should be brought into such position that there will be no overstretching, and atrophy should be guarded against by means of massage, electricity, and hot-air baking. Care should be exercised in baking on account of the diminished sensibility, which might lead to severe burns.

The prognosis in nerve suture depends upon the type of operation, the after treatment, and particularly upon the location of the injury. Plexus injuries and injuries of the more central portions of nerves offer in general a much more unfavorable prognosis than the more peripheral injuries.



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